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INFLAMMATION OF THE URETERS IN THE FEMALE.[1]

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It is for the purpose of calling attention to a disease about which little has been said and written, and especially to its causation and treatment, that I have prepared this paper. I am convinced that ureteritis is common, that it is often overlooked, and that it is mistaken for other conditions in no way connected with it except in location.

Frequency.—My knowledge of this disease extends over only a few years, dating from a paper by Dr. H. A. Kelly,[2] in 1888, and a personal visit to him, in which he kindly taught me to recognize the ureters by palpation. Since that time I have met with many cases—some of them recent, but a larger number of many years' duration. Some of them I have seen only in consultation and have no knowledge of their progress; others I have followed for a longer time.

From the number met with, I am convinced that inflammation and irritation of the ureters is exceedingly common, and that its general recognition is a matter of great importance. As yet the text-books are almost silent on the subject, the latest—Garrigues' excellent work—not mentioning it. In fact, the literature on the subject is exceedingly scanty.

In order to prepare for what I have to say, I must first refresh your memories regarding the anatomy of these important ducts.

Anatomy.—The ureters are two delicate tubes leading from the pelvis

[1] Read before the American Gynecological Society, Washington, D. C., May, 1894.
[2] "Relation of the Ureters," Gynecological Trans., 1888.

of the kidney to the bladder. They run nearly parallel as far as the brim of the pelvis. After entering the pelvis they run downward, backward, and outward to the side of the pelvic wall, to a point near the spine of the ischium, where they hug the bone closely. Then, reversing their course, they come nearly together where they enter the bladder. In length they never exceed fifteen inches, averaging between ten and twelve inches (Van Hook). The point opening into the bladder is about three-quarters of an inch in front of the cervix; the points of opening are from one to one and one-half inches apart, and one inch from the neck of the bladder.

Van Hook[1] calls attention to the fact that the ureter has three points of diminution of calibre, which may give rise to mistakes in the search for pathological stenosis. The first is between one and one-half and two and one-half inches from the pelvis of the kidney, according to Dr. Tangreary's[2] measurements. The second is at the junction of the pelvic and vesical portions, and the third (in three out of five) is where the ureter curves over the iliac artery.

It is in the latter part of their course, where they pass under the base of the broad ligament and through the bladder wall, that they can best be recognized by the examining finger. An exact appreciation of their position in the pelvis is, therefore, necessary before we can expect to study them. By palpation they can be recognized, especially when enlarged by disease, as two cords, running from a point a little in front of the uterus and a little to one side of the median line, directed at first toward the sides of the pelvis, and then curving somewhat sharply backward until they go beyond our reach near the spine of the ischium. In front of the uterus their ends are separated by an inch or a little more of space. Tourneur[3] claims that, when enlarged by disease, they can be distinctly palpated through the abdominal wall above the pelvis.

That the ureters may be the seat of disease has long been recognized. The causes and nature of their diseases are only now beginning to be studied and the diseases to be regarded as independent pathological entities.

Unquestionably the most common disease is inflammation, and I have learned by observation to recognize seven causes for this trouble.

CAUSES OF INFLAMMATION:

1. Injuries during childbirth.
2. Previous disease of the bladder.
3. Gonorrhœa.
4. Suppuration of the pelvis of the kidney.
5. Pelvic disease, such as pelvic peritonitis, cellulitis, and tumors.

[1] W. Van Hook: "Surgery of the Ureters," Journ. Amer. Med. Assoc., December, 1893.
[2] Quoted by Van Hook.
[3] C. Tourneur: De l'Uréterite et de la Péri-uréterite. Paris, 1886.

6. Abnormal conditions of the urine.

7. Tuberculosis.

1. *Injuries from childbirth.* In our *Transactions*[1] for 1890, Dr. Skene called our attention to the first of these causes and the resulting effects. During the last winter I met with several cases. In each there was a chill, with high fever, pelvic pain, and vesical irritation, coming on several days after labor. The labors were none of them especially severe or long, but were all in primiparæ. A careful pelvic examination showed nothing wrong with the uterus, tubes, or pelvic peritoneum; but pressure over the lower end of one of the ureters showed great tenderness. After the third or fourth day an examination of the urine revealed an acid reaction with a large amount of pus and some blood. There was no mucus or other evidence of bladder trouble. As Skene points out, bimanual manipulation over the kidney on the affected side showed some tenderness.

In none of these cases was there a history of any pre-existing disease of the bladder or kidney. In the first case I gave a grave prognosis; but the patient recovered, as did the subsequent cases. Skene met with one fatal case.

Skene considers that the cause is to be found in pressure on, or injury of, one or both ureters by the child's head or the blade of the forceps, the pendulum movement of the forceps being particularly prone to produce trouble. Especially is it likely to occur if the head pushes down an undilated cervix and the bladder with it.

A most instructive account of an autopsy is to be found in the article referred to.

Besides these more serious lesions, I have noticed an irritation lasting a long time, and evidently started by the pressure of the child's head, but kept up by other causes. I will here cite one illustrative case:

I delivered Mrs. A. of a large child some years ago with forceps. The head was R. O. A. Following the labor she had a mild attack of sepsis, due to the retention of a small bit of very adherent membrane, from which she fully recovered. Soon after the labor she complained of a frequent desire to urinate, and it was necessary to pass the catheter quite often. This symptom seemed to persist, and her family physician tells me that he treated her for "irritable bladder" for a long time.

A few months after her recovery she was in one of our large cities; and, as her bladder began to trouble her again, she consulted one of our distinguished Fellows. He sent her back to me, advising that I sew up very slight tears in the cervix and perineum in hopes of relieving, as he said, the tenderness in the right broad ligament. (Cellulitis?) On examination, I found the right ureter very tender, and the urine quite acid. I gave her large doses of alkalies, combined with an alkaline mineral water, and in a few weeks she was entirely relieved of the distressing symptoms.

[1] A. J. C. Skene: "Injuries of the Ureters during Labor," Gynecological Transactions.

Undoubtedly the trouble began in childbed, from the pressure of the head (R. O. A.) on the right ureter, and was only relieved when all external irritation—the hyper-acid urine—was removed.

2. *Previous bladder disease.* In certain deformities of the bladder, such as fissure, the ureters are generally greatly dilated. In cystitis, except in the gonorrhœal form, extension to the ureters either does not occur or is a late manifestation. If a neoplasm or hypertrophy of the bladder obstruct the exit[1] of the urine from the ureters, dilatation of the ducts gradually takes place. "When the ureteric openings are dilated so that urine regurgitates at each vesical contraction," infection of the ureters is sure to follow with serious results.

Some authors have thought that all diseases of the ureters have their origin either in the bladder or the kidney, the ureteritis being called "ascending" or "descending," as the case may be. It has never been my fortune to see a case of ascending ureteritis, except when it has had a gonorrhœal origin. Still it may be possible that an ordinary acute septic cystitis may be conveyed to the ureters. But, if this be so, it must —in the female, at least—be rare. Coe[2] mentions having seen two cases of pyelitis follow cystitis. The course of the infection was presumably by the ureters.

3. *Gonorrhœa.* That gonorrhœa may extend to the bladder is generally acknowledged, though gonorrhœal cystitis in women is either by no means common or does not often attract attention. If gonorrhœa once reaches the bladder, the inflammation may also extend into the ureters. It is possible that after the disease has subsided in the bladder it will still lurk in the ureters, either because the conditions are more favorable, or because, the bladder symptoms attracting notice, treatment is directed to the bladder but does not reach the ureters.

I have seen several cases in which I am certain that this had occurred. There was a direct history of gonorrhœa, and other evidence in the presence of inflamed and suppurating Fallopian tubes. I have no doubt that many of the failures to obtain entire relief from pain after cœliotomy for pus tubes are due to coincident but unrecognized gonorrhœal ureteritis.

4. *Pyelitis and pyelonephritis.* Suppuration of the pelvis of the kidney and of the kidney itself is more often, in my belief, due to previous disease of the ureters than *vice versa*. Still, many cases are recorded where the disease is supposed to have been due to emboli or other trouble starting *de novo* in the kidney or pelvis. Unfortunately, in the many recorded cases the condition of the ureters is seldom mentioned. A calculus formed in the pelvis of the kidney frequently leads to suppuration; and with almost absolute certainty, the ureters will

[1] Skene: Diseases of the Bladder and Urethra in Women, p. 242.
[2] "Deaths from Visceral Affections after Ovariotomy," Gynecological Transactions, 1889.

then, sooner or later, be involved. This is the so-called descending ureteritis. It is my experience that most of the cases where the trouble begins in the kidney or its pelvis are either due to calculi or tuberculosis.

5. *Pelvic and uterine disease.* The relations existing between uterine and pelvic diseases and diseases of the ureters have been carefully worked out by Engelmann.[1] With many of his conclusions I disagree *in toto.* Time fails me to carefully criticise his paper, and I shall only allude to a few points of difference, while quoting many points of agreement.

I have very little faith in the extension of inflammation from contiguous structure, except along surfaces. I do not believe that ureteritis ever follows an inflammatory action in the tubes, ovaries, pelvic peritoneum or parametrium by direct extension. If the two coexist, I would look either for a common cause in a gonorrhœal infection, or the ureteritis may be due to the inflammatory exudation causing pressure and obstruction.

Still less can I believe that an anteflexed fundus can by pressure, even for a very long time, and "under conditions favoring the progress of the disease," induce a ureteritis, pyelitis, and pyelonephritis.

I wish to enter my protest against what I believe is a common mistake. An anteversion or anteflexion is found, and with it a degree of vesical irritability, which is attributed to the pressure of the uterine body on the bladder. It seems to me to be ridiculous to suppose that an organ which weighs only one and one-half ounces, one-half of which is supported by the vagina, can produce so much trouble by its weight alone. Especially is this so when we see the bladder pushed out of place and pressed upon by tumors without any special symptoms of this kind. Under such circumstances, I have never failed to find some other condition to account for the vesical irritation, usually an abnormal condition of the urine.

Pressure on the ureters is recognized by all writers on this subject as a frequent cause of ureteral disease. That a sufficient degree of pressure may be made by a firm neoplasm, such as a uterine fibroid or ovarian tumor, cannot be doubted. In cancer also the disease itself, or the zone of infiltration which surrounds it, may directly press upon and even occlude a ureter. Obstruction from any cause may lead to ureteritis, pyelitis, and its consequences. In ulcerating cancer, if the disease opens the ureter, direct infection, with all its results, may take place.

Engelmann holds that "above all, retroflexion and prolapsus uteri" may cause pressure on the ureters. As far as retroflexion is concerned, I have never seen any clinical evidence that this may occur; nor does it seem likely from the anatomical arrangement of the parts. The ureters

[1] "Renal Disease following Utero-ovarian Lesions," Gynecological Trans., 1889.

are so far separated, directly behind the cervix, that there is plenty of room for an enormously enlarged retroflexed fundus between them. Where they pass over the brim of the pelvis they are well protected by the promontory of the sacrum, and are, moreover, too high for a retroverted uterus to reach them. Nor is the weight of the uterus enough. In prolapse, the course of the ureters may be interfered with, but in the many cases of prolapse and procidentia that I have met with I have never seen anything to support this conclusion.

It is in Engelmann's third class that it seems to me the most evident connection between diseased pelvic viscera and diseases of the ureters is to be found. He claims that "functional derangement and finally morbid changes are produced by nervous influences emanating from the diseased pelvic viscera."

This may be brought about in two ways: "as reflex phenomena," or "by perverted action of the secretory nerves, due to the intimate connection of the uterine and renal plexus."

My interpretation of the phenomena would be as follows:

In the first place, it is well recognized that the stomach and other digestive organs bear a very close relationship, as regards their functional activity, to the sexual organs. Now, if the digestive processes are disturbed by primary disturbances in the pelvic viscera, the resulting indigestion will cause abnormal conditions of the urine. It is in this way, as I shall explain later, that I believe inflammation of the ureters is generally brought about.

Perverted action of the nerves governing the renal function often results in a deficient amount of urinary excretion. This condition, generally spoken of as "renal insufficiency," is, I believe, often a result of a stimulation of the inhibitory influence of the nerve centres, due to peripheral irritation in the sexual organs. It is often found associated with ureteritis, and, I am fully persuaded, often stands in the relation of cause and effect.

6. *Abnormal conditions of the urine.* It is my belief that abnormal states of the urine cause the great majority of cases of ureteritis. The urine which is at the bottom of the trouble is excessively acid, often depositing a thick sediment on standing. It is scanty and high-colored; or this condition may alternate with a profusion of pale limpid urine of very low specific gravity. The constant passage of this abnormal urine irritates the whole urinary tract, and is, I am fully convinced, the most common cause of inflammation of the ureters.

This particular kind of urine will at once be recognized as that characteristic of the condition often known as lithæmia. To detail the causes of lithæmia is beyond my limits.

One point I must mention again, and that is renal insufficiency. In many of these cases I have found the total amount of urine reduced to

eight or ten ounces in the twenty-four hours. Such urine is often of low specific gravity, showing a great deficiency in the total amount of solids excreted. It is generally acid and contains a variety of crystals. The recognition of this condition—whether we consider it only as one factor in lithæmia or as a separate affair—is of the utmost importance for the successful treatment of these cases.

Renal insufficiency being often the result of reflex action, as already explained, we must look for the exciting cause; and this we may find in the pelvic organs or in some distant organ, as, *e. g.*, in the eye. If it be in the pelvis—or wherever it may be—we shall make little progress in the management of our case until we recognize the cause.

I have noticed that some kinds of fruit seem to cause a state of the urine which is particularly obnoxious to the urinary mucous membranes. Strawberries are especially noticeable in this respect.

7. *Tuberculosis.* Tuberculosis as a cause of disease of the ureters has been occasionally recognized.

The case of a young girl came under my notice a year or more ago. The ureters were enormously enlarged, and there was much pus in the urine. I turned her over to Dr. Chas. G. Stockton for general treatment, and Dr. Bergtold found tubercle bacilli in the urine. She later went to New York, where her kidney was opened, and a condition was found which was called tubercular pyelitis. A few months ago Dr. Roswell Park removed this kidney, finding numerous foci of pus and other evidences of tubercular disease.

It may be questioned whether tubercular disease of the ureters exists independently of the same disease in the kidney.

Pathological Anatomy.—I have had no opportunities of studying this disease post-mortem, and have not had access to any work treating fully the pathological anatomy of the ureters. Judging from what I have observed clinically, and from the reports of cases by other observers, I think we may distinguish several forms or stages of ureteritis.

First, the catarrhal form, in which there is a little swelling of these tubes with desquamation of the epithelial lining. It is my belief that in slight cases, judging from the evidence gained by examination as well as from the symptoms, the force of the disease is first spent on the lower end of the ureter, especially the part in front of the broad ligament.

In other cases the surface of the tubes seems to give forth a plentiful purulent secretion, which indicates an ulcerated or granulating condition of their lining membranes. Tourneur says that when these ulcerations occur a thickening in the surrounding connective tissue takes place with, perhaps, adhesions of the peritoneum, giving an irregular outline to the course of the ureters.

Sometimes the tube is greatly thickened by inflammatory deposits in the walls. This may reach a point where the ureters are as large as a

lead pencil, or even larger. In the case of obstruction, dilatation, even to an extreme degree, may take place, accompanied by a certain amount of thickening. Tourneur likens these dilated and thickened ureters to the arteries in a cadaver.

I have now under observation a case in which the ureters are greatly thickened, and in which the right one seems to be dilated or sacculated just behind the broad ligament.

The pelvis of the kidney is doubtless generally more or less involved with the ureters; but that it is always so is not proven. A physical examination in several cases of so-called "pyelitis" has shown that the ureters were the parts chiefly involved. That these conditions may end in involvement of the pelvis and of the kidney itself, I have had clinical evidence. In several cases perinephritic abscesses have developed; and in two abscess of the kidney has developed, as proven by operation.

Usually both ureters are involved in the pathological processes; but often one side—usually the left—is much more seriously affected than the other.

Coincident Affections of Other Organs.—The bladder is rarely directly affected by coincident inflammation; that is, there is seldom a general cystitis. But in a case recently examined, in which the pain and tenderness are confined to the left side, the surface of the bladder around the mouth of the ureter, as shown through a Kelly speculum, was quite red and congested. The urethral orifice on this side is only half the size of that on the unaffected side.

In another case there was a patch of granulations around the orifice of the right ureter as large as a twenty-five-cent piece. The possible existence of stricture at the point of opening is something to be considered.

It is a matter for future investigation whether the vesical tenesmus is not always due to some localized congestion, granulations, or ulceration around the mouth of one or both ureters on the bladder wall. In consequence of the constant emptying of the bladder its walls may become contracted and almost incapable of expansion. This fact must be taken into consideration in the treatment, and means taken to overcome the contraction.

Other complications are the various forms of tubal, ovarian, and uterine disease. Endometritis is the commonest. Retroversion and prolapse are not uncommon. But in a considerable number of the cases met with the sexual organs have been only slightly affected.

It will be at once admitted that some of the conditions described as producing ureteritis will also in time produce changes in the kidney, especially contracted or granular kidney. This makes the disease a more serious one. Whether there be any relation between floating, or movable, kidney and ureteritis I am unable to say, never having met with a case in which the two conditions coexisted.

Late in the disease, abscesses, either in or around the kidney, or else around the ureter, may form. I have seen several instances of perinephritic abscess, and one which I think had its origin in the cellular tissue around the ureter. It pointed in the iliac fossa. As already said, Bright's disease may come to end the scene.

Symptoms.—The most constant symptom is frequent micturition, which may even become continuous. In only a very few cases with which I have met has this symptom been absent. The desire is most frequent during the day, but the rest is often broken by calls to empty the bladder at night. In the worse case I ever saw, when I was summoned the woman had been sitting on a commode for a month, night and day, only occasionally snatching a moment's sleep. The desire to urinate was constant.

In this case the ureters were as large as lead pencils and exquisitely tender; there was pus in the urine, but the bladder seemed to have the usual tolerance, that is, there was no special pain on distending it with fluid. Relief was obtained, however, by the establishment of a vesico-vaginal fistula.

The next symptom in importance is pain over the ureters—one or both—the left side being more commonly affected than the right. This pain is described as burning or boring, and is nearly constant, but is almost always greatly aggravated as the menses approach, even becoming agonizing during the flow. This peculiarity has often led to an error in diagnosis, the disease of the ureters being mistaken for ovarian disease. Several cases have come to me to have the ovaries removed, when all the trouble lay in the ureters. One case in particular has been examined by several excellent men, all agreeing that the ovaries were the seat of the intolerable menstrual pain, and ought to be removed. When I last saw the woman she still possessed her ovaries and was perfectly well.

One curious symptom I have noted as frequently existing in cases associated with renal insufficiency, *i. e.*, an absolute distaste for water. I have frequently told the patient, without asking her, that she did not drink water enough, and she has replied that she did not drink any. In some cases water seems actually to produce nausea. This distaste for water is often very hard to overcome.

Should cystitis coexist, the symptoms of this disease may entirely mask the other. Again, should the kidneys become affected a new train of symptoms will arise. In lithæmic patients other manifestations of the condition may develop. Many of these patients are subject to bilious attacks and sick headaches. Gastric and intestinal dyspepsias are common, and there is often a history of inherited rheumatism or gout. Great depression of spirits is not an uncommon symptom. Even in the lithæmic cases the symptoms often seem to date from childhood.

Sometimes the disease is intermittent, attacks coming now and then; but usually it runs an essentially chronic course. I have met with cases which had lasted for twenty years, gradually getting worse, until life was very nearly unendurable.

The pain is sometimes steady, and again it is spasmodic. It may be stationary over the ovarian region, or it may be felt over the hip and down through the iliac fossa. In some cases the pain is intensified by walking; others are unable to ride in any vehicle, the jar and movement causing much pain.

Attacks of gravel or the passage of small stones from the pelvis of the kidney are rather common, and may be distinguished by the usual symptoms. In some cases the passage of plugs of inspissated pus produces symptoms similar to those of gravel.

DIAGNOSIS. This disease may be recognized, first, by the symptoms enumerated; secondarily, by the physical examination; and, thirdly, by an examination of the urine.

It is needless to go over the symptoms again; only it must be remembered that they do not give conclusive evidence. As already stated, the symptoms may be mistaken for those of ovarian, tubal, and uterine disease, on the one hand, and for those of the bladder and kidney on the other. It must be borne in mind that frequent micturition is not always present.

It is on the physical examination, then, that we must place the chief reliance. That the ureter is often easily palpable has been clearly demonstrated. Pawlik, H. A. Kelly, and others have, by their teaching and writing, established this fact conclusively. In fact, there is nothing easier than the palpation of an enlarged ureter in the pelvis. When it is not enlarged the matter is not so easy; and I am convinced that it is not possible to perfectly and clearly distinguish the ureters in all women. If, however, we know their anatomical position, and pressure over these points—and over these points alone—elicits pain, we may, if the symptoms are corroboratory, safely infer that we are pressing upon the ureters, and that they are diseased. Care must be taken not to confound tender spots in the other structures of the pelvis, so frequently discoverable, for the ureters.

METHOD OF PALPATION.—*Pelvic.* The palpation of the ureter in the pelvis is done as follows: The finger is carried along the anterior vaginal wall upward and outward near the brim of the pelvis to one side of the uterus. It is then passed forward, stroking the pelvic wall and carefully feeling for a cord-like body under it. Sometimes a bimanual examination will greatly aid in discovering the ureters. Anybody who has once distinguished an enlarged ureter will be astonished at the ease with which it may be done.

In every case that I have seen, tenderness on pressure was present,

and sometimes to an extreme degree. A feeling of a desire to urinate is often complained of on touching the ureters; and Kelly describes a case in which micturition took place over his hand while examining.

By tracing the course of the ureters carefully with the finger-tips, disease of the bladder and urethra may be excluded. Should bladder tenderness coexist it will be hard to distinguish the ureteritic disease, unless the ureters are enlarged, or at least distinctly palpable.

Abdominal palpation. As already mentioned, Tourneur[1] lays great stress on the palpation of the ureter through the abdominal wall. He says: "At the level of the superior strait the ureter can be found at one-third the distance which separates the anterior superior spines of the ilium."

In women who have borne children and have, in consequence, a relaxed abdominal wall, he claims that it is astonishingly easy to palpate the enlarged and thickened ureters. If there be a peri-ureteritis, the investigation is further facilitated; and in some cases irregularities or swellings are noted along the course of the ureters. If there be simple dilatation without hypertrophy, palpation is more difficult. He considers this as the principal method of determining changes in the form and consistence of the ureters, and consequently of the utmost importance. He cautions us to be careful not to mistake a chain of enlarged glands (the ilio-lumbar) for the enlarged and irregular ureters. These glands are especially likely to be affected in tubercular disease.

His method would seem to supplement the pelvic palpation. For its performance it is necessary that the bowels should be thoroughly emptied, and that the patient should lie on a hard table with the knees drawn up to relax the abdominal walls.

I have never practised abdominal palpation of the ureters.

EXAMINATION OF THE URINE.—This is an absolute necessity, both for diagnosis and as giving important indications for treatment. No examination can be considered as complete unless it embraces an estimation of the amount of urine passed in twenty-four hours, as well as a careful chemical and *microscopical* examination. Many practitioners consider that when they have tested for albumin they have done their whole duty. Such a test is practically useless, showing nothing regarding the abnormalities to be met with in ureteritis.

The urine will often be scanty—I have seen it as low as six ounces in twenty-four hours; always acid in reaction, often hyper-acid, unless cystitis coexists, and often of low specific gravity, 1010 to 1015.

The sediment will consist of urates, uric acid, calcic oxalate, often pus, and a little epithelium. All attempts to locate the seat of the disease by the presence of peculiar types of epithelial cells will fail. In

[1] Loc. cit.

old cases, where pus is present in large amounts, no epithelium will be found. Blood-cells in greater or less proportion will often be found with the pus.

The amount of mucus in the urine will be very slightly increased. There are no mucous follicles in the pelvis of the kidney or ureters.

As the bladder is abundantly supplied with such follicles, the presence or absence of mucus is of much diagnostic value. Urine containing pus without mucus and acid in reaction is, I believe, a sure indication of inflammation in the urinary tract above the bladder—leaving out, of course, the presence of an abscess opening into the bladder.

On the other hand, alkaline (ammoniacal) urine with mucus and pus is a sure sign of cystitis, but does not exclude involvement of the ureters or pelvis of the kidney.

Palpation or catheterization of the ureters is then the resource for deciding whether the ureters are also involved. The presence or absence of albumin may be of some significance. I have never found anything more than the faintest trace of albumin in purulent ureteritis; generally there is none.

The presence of albumin with pus may mean, then, either that the kidneys are affected or that the pus comes from an abscess cavity, the liquor puris furnishing the albumin. The further history of the case will decide which.

It is by no means necessary that the ureters should be much thickened before pus is produced. I have seen it in comparatively recent cases where the ureters were not at all enlarged, but only tender. Under proper treatment these cases rapidly recover.

Prognosis.—The prognosis in a given case of inflammation of the ureter depends entirely upon the stage in which the disease is met with and upon the condition of the ureters as recognized by palpation. If the trouble has not lasted very long, and the ureters are not much thickened or enlarged, the prognosis is good, though the time necessary for a cure will be considerable. In old chronic cases, where great thickening of the ureters has taken place, and where large amounts of pus are found in the urine, the outlook for the patient is certainly not good. It is altogether likely that abscesses will form in or around the kidney, and that the patient will succumb from them, or later from some form of Bright's disease.

Nevertheless, I have seen one case, which I have followed now for nine years, and which has apparently recovered. The trouble began, I infer, with lithæmia and a mild ureteritis, under my care. This was entirely unrecognized and not properly treated. The ureters gradually got thicker; and when I examined her again, some four years ago, I found them moderately enlarged, the disease having then, judging from the symptoms, lasted about five years. Later, she had an abscess of

the right kidney, which was opened and drained, and which left a sinus. Finally the kidney was removed, and the patient is, I understand, now perfectly well. The other ureter, however, is slightly thickened, and may ultimately give trouble.

As already stated, I have seen cases which have lasted twenty years, judging from the symptoms, in which there was only moderate thickening and a small amount of pus in the urine. Other cases run a much more rapid course. It will thus be seen that a prompt recognition of these cases and a proper treatment is of great importance.

TREATMENT.—The treatment may be considered under the heads:

1. Constitutional.
2. Local.
   a. Through the urine.
   b. Direct local treatment.
3. Surgical.

1. *Constitutional.* From what has been said regarding the causation of this disease, it will be at once seen that constitutional treatment is of the utmost importance. Unless this is carefully carried out we cannot expect to get a normal condition of the urine, which is absolutely essential, not only to prevent the further progress of the disease, but also to favor a cure.

To this end the patient should be put in the best possible environment; have perfect freedom from mental worry and anxiety, in a situation where she can have plenty of outdoor air and exercise, and where everything shall be done to promote the general health. A careful regulation of the diet is absolutely necessary, even where the case be of gonorrhœal origin; for the urine must be kept bland and unirritating under all circumstances. In many cases sugar and starches seem to be very poorly digested. They thus do harm, and are to be avoided. Alcoholics, especially wine and beer, must be given up.

In cases where the kidneys are not acting freely, or where there is marked renal insufficiency, the use of hot-air baths—either the regular Turkish bath, or, better still, the bath given with an alcohol lamp placed under the chair, the patient being swathed in blankets—will produce good results. By this means the skin is made to do duty for the kidneys, and the poisonous excretions which have been retained in the blood are gotten rid of. Moreover, the dry hot-air bath acts as a derivative, drawing the blood to the surface, thus lessening the congestion of the internal organs. Under this treatment the kidneys will quickly resume their work, and the amount of urine will be increased very markedly, notwithstanding the profuse sweating.

Massage is also of great use in many of the cases, and can be used directly after the hot-air bath.

Under all circumstances, but especially where there is renal insuffi-

ciency, I advise the free use of water. The psychical effect is often better if we use a mineral water; but I do not believe that any of the waters in the market contain a sufficient amount of alkali to do very much good. It is the water, and not so much the mineral ingredients that it contains, that accomplishes the result. I have very little faith in diuretics.

The use of alkalies is of the utmost importance, and they should be used long and persistently, in sufficient quantity to keep the urine alkaline, or at least neutral. For this purpose I have used the liquor potassæ in doses of five drops and more; also the acetate and citrate of potassium, and Rochelle salts and bicarbonate of soda. In many instances the salts of lithium are of great value. Etheridge[1] advises lithium combined with potash to increase the flow of urine. In some cases one agent, and in others another, acts. A little trial will prove which has the best effect with each particular patient.

Agents which act upon the liver, especially where there is constipation, are also very useful. To this end I have used podophyllin, leptandrin, euonymin, and other agents of this nature. The greatest care should be taken to see that the bowels act freely. Where there is a history of constipation, flushing of the colon is of inestimable value.

If anæmia exists, the use of iron and arsenic will be beneficial.

It is not necessary for me to enlarge further upon this matter, only to insist that everything must be used to restore the patient's general system to normal condition. In several instances of comparatively recent cases, other means having failed, I have seen the greatest amount of good come from an exclusive milk diet. This must be kept up until the symptoms are entirely relieved and the pus disappears from the urine. I have seen a number of patients entirely cured in this way.

2. *Local. a. Through the urine.* It is well recognized that certain agents are excreted through the urine and have an influence upon the membranes over which they pass on their way out of the body. The various agents ordinarily used in gonorrhœa, such as copaiba and sandalwood oil, have in several instances proven beneficial, especially where there is a great deal of pus in the urine. Sandalwood I have found especially valuable.

I have also tried carbolic acid, salol, and hydronaphthol, with the idea of getting their antiseptic action upon the ureters, or their germicidal action in the urine as they pass out. I cannot say that I have seen any great results from the last-named group; in fact, we are greatly in need of a drug which, when given by the mouth, will produce an antiseptic action in the urine. Benzoic acid, perhaps, comes as near as anything; but, as it produces an exceedingly acid state of

[1] Personal communication.

the urine—hippuric acid being excreted—it does not seem exactly indicated. Its beneficial effects in cystitis with alkaline urine is well known; but I have not seen the same results in ureteritis.

Tourneur praises the essence of turpentine in capsules, two to six, or even eight, a day. He considers that turpentine renders the urine less irritating and at the same time notably increases the amount.

b. Direct local treatment. The discoveries and inventions of Dr. Howard A. Kelly [1] have opened up a new field for the local treatment of diseases of the ureter. By his method it is now in the power of everyone to pass an instrument into the ureters with comparative ease. An operation which was formerly considered very difficult, even for an expert, is thus brought within the reach of all; and, though sufficient time has not yet elapsed for us to have formulated any particular plans of treatment, or to have tested any special drugs, still the general principles of treatment of inflamed mucous membranes may be applied here. I have recently dilated and catheterized the ureters through Kelly's speculum, and am about to institute a series of experiments in the action of various drugs. Of course it will be necessary to go slowly, as we have no means of knowing as yet what the susceptibility of these structures is to the different agents. I propose to try injections of boric acid, weak solutions of nitrate of silver and so on, and see what can be accomplished in this way.

3. *Surgical treatment.* Much has been written lately in regard to the surgery of the ureters. As yet, however, very little has been done for the application of surgery to their treatment. Personally, I have no experience with anything of this nature further than catheterizing and dilating the ureters. This I have done a few times, and from it alone have never seen any great results, although one case where I dilated the ureters with the catheter passed through the bladder was very much relieved of her pain for a considerable length of time. After about a year the old trouble returned; and before the patient could come to me for a repetition of the operation she died.

TREATMENT OF THE BLADDER.—In one case—the one which I noted on a previous page as having had almost continuous *ardor urinæ*—I procured great relief by making a permanent vesico-vaginal fistula. After that was done, the patient was able to leave her commode, to lie down and sleep well. She died, however, of an abscess which started either around the kidney or outside of the ureter, and opened in the iliac fossa.

In another case in which I opened the bladder, the ureteritis being at that time unrecognized, no relief followed. The patient afterward put herself under the care of a very distinguished member of the

[1] "Direct Examination of the Female Bladder, etc.," Am. Journ. Obstet., January, 1894.

American Gynecological Society, but received no benefit, and, I think, ultimately committed suicide, owing to the severity of her symptoms.

If the bladder be much contracted and very irritable, benefit can often be obtained by distending the bladder with fluids. For this purpose, weak salt solutions can be used, or solutions of boric acid, or other similar agent. If there be granulations around the ureteric openings, congested areas or ulcerations, they can be treated topically by the aid of Kelly's speculum. I have recently scored a brilliant success in the local treatment of such a case.

From what has already been said, it will be seen that much more research is necessary before we can arrive at a truly satisfactory treatment of this affection. If, however, I can succeed in rousing the attention of the profession to its existence and to the necessity of recognizing and treating it, I think that the beginning of the end will have been reached.

CERTAIN CASES OF HETEROTOPIA OF WHITE MATTER OCCURRING IN THE HUMAN MEDULLA OBLONGATA.

By James D. Heard, M.D.,
of Pittsburg, Pa.

(*Communication from the Laboratory of Prof. Obersteiner, in Vienna.*)

In the extensive literature of the human central nervous system but little mention is made of the presence of congenital histological anomalies in the cord and medulla oblongata. In the cord, Van Gieson[1] could find mention of but four cases of true misplacement of gray matter, while no cases of heterotopia of white matter had been published. In the medulla oblongata anomalous bundles of white matter had been observed in three instances. To these observations Van Gieson added a case of his own, and Schaffer[2] has since added a fifth. If with these be included the bundle described by Henle[3] as an occasional anomaly, but afterwards[4] identified by him with the respiratory fasciculus, the number of cases rises to six. These six comprise the only genuine cases of heterotopia of white matter in the medulla of which I can find record. The cases are as follows:

Case I.—In the twenty-first volume of the *Archiv für Psychiatrie*, Prof. Pick[5] reported the observation of an anomalous bundle of nerve fibres occurring unilaterally in a human medulla oblongata. It commenced at the level of decussation of the pyramids as a small, sharply circumscribed collection of nerve fibres originating from the remains of the lateral column, and occupying a position anterior and somewhat

mesial of the head of the posterior horn. The fibres comprising the column were easily distinguished from those surrounding it by reason of their larger calibre and consequent darker stain (Weigert method as modified by Pal). As the bundle ascended its diameter increased and arching fibres appeared, which separated it from decussating fibres and gray substance. At the level of the upper third of the olive its component fibres separated, and could be followed into the corpus restiforme; while at a somewhat higher level the anomalous structure had entirely disappeared. At no part in its intermediate course could Pick trace any connection between the fibres composing the bundle and surrounding structures. He can offer no explanation as to the physiology of the bundle, but regards it as an abnormal path for the fibres which the corpus restiforme always receives from the lateral columns. He identifies it with the bundle of Henle mentioned above, which is described in the first edition of the *Handbuch der Nervenlehre* as "one or as two cylindrical and sharply circumscribed nerve-like cords of occasional occurrence, and having a diameter of 25 to 50 mm., always occurring unilaterally, and situated in the posterior boundaries of the reticular substance, anterior to the head of the posterior horn." As Henle subsequently identified this structure with the respiratory fasciculus (*Handbuch der Nervenlehre*, 2d edition), Pick's observation possesses the merit of a rediscovery.

Case II.—Cramer,[6] in the examination of the central nervous system in a case of cerebellar hemiatrophy, discovered an anomalous bundle of white fibres situated upon the side of the lesion, and corresponding in size, cross section, and location with the bundle of Pick. It arose at the level of decussation of the pyramids, and reached its greatest development at the sensory decussation. Here it was seen as a nerve-like cord, completely surrounded by arching fibres, and having no demonstrable connection with surrounding structures. At the lower third of the olive the column had subdivided into two, and fibres were observed to leave it and radiate in the direction of the corpus restiforme. That these fibres really entered the restiform body could not, however, be demonstrated. Although the exact origin and the destination of the fibres was not determined, the column was regarded by Cramer as identical with that of Pick, and he reached the same conclusion as to its anatomical significance—namely, as an abnormal path for communicating fibres between lateral column and corpus restiforme, and possibly cerebellum.

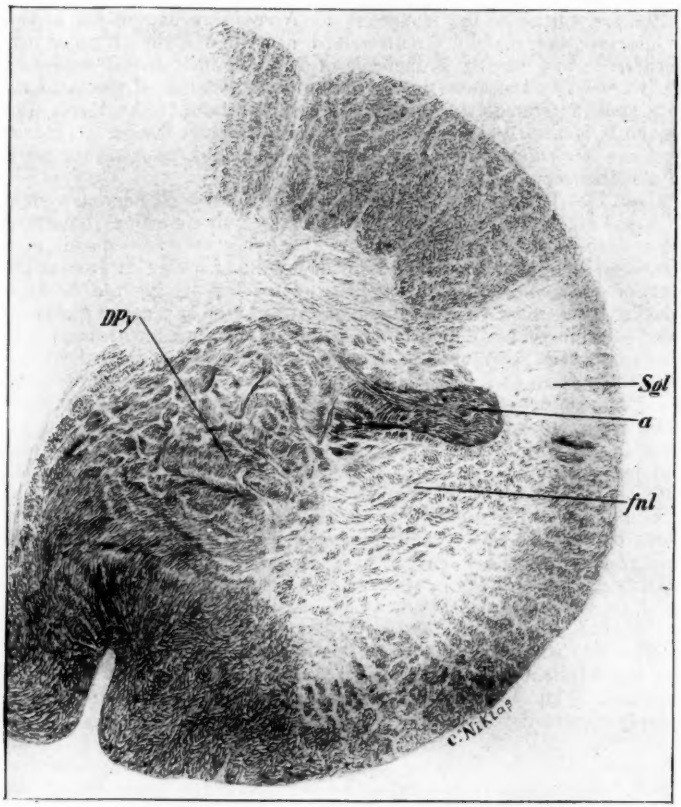
Case III.—Schaffer,[7] in the *Neurologisches Centralblatt* for August, 1890, reported the observation of an abnormal fasciculus which arose at the upper level of the decussation of the pyramids, and was situated internal and posterior to the gelatinous substance of Rolando, exactly at the angle which the posterior horn forms with the nucleus funiculus cuneatus. The cross section of the bundle was elliptical, and it was sharply circumscribed anteriorly, internally, and posteriorly by arching fibres. Internally the fibres composing the bundle had a longitudinal direction, while the fibres of the outer half had an oblique course, which, however, tended to become longitudinal as the fibres neared the centre of the bundle. Externally they could be traced to the cells of the nucleus funiculus cuneatus. Although the column was seen to deviate toward the respiratory fasciculus at the level of the lower third of the olive, no communication with this structure was demonstrable.

At a somewhat higher plane the bundle had disappeared, its fibres having entered the corpus restiforme.

Although the location of the bundle is posterior to that of Pick's, and its fibres appear to originate in the posterior instead of the lateral columns, Schaffer identifies the two structures. He regards the bundle as an abnormal path for those fibres which the corpus restiforme receives from the posterior column.

CASE IV.—Kronthal,[7] in a case of bulbar paralysis, discovered two anomalous unilateral bundles of white fibres lying mesial of the hypoglossus nucleus, near the floor of the fourth ventricle. After thirteen sections had been cut the bundles united and ganglion cells appeared.

FIG. 1.



Transverse section at lower level of decussation of pyramids.

Sgl, substantia gelatinosa. *fnl*, Lateral column; dark fibres separate from those engaged in the decussation, and proceed laterally, to collect at the inner border of the substantia gelatinosa as a distinct round bundle, *a*.

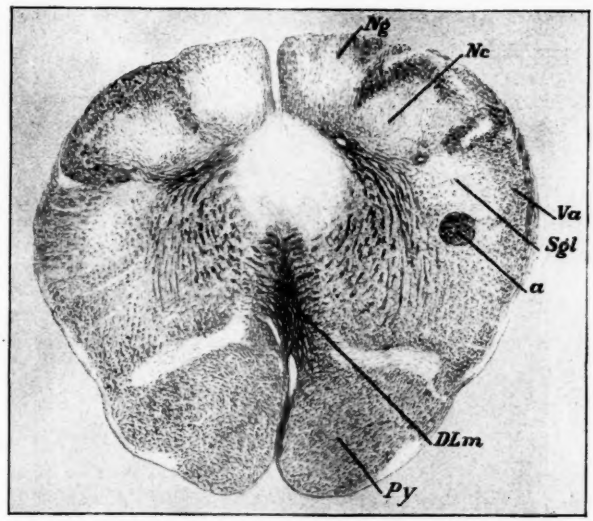
The origin of the bundles could not be determined, and after the appearance of the ganglion cells they could no longer be traced.

CASE V.—In the examination of a human medulla, Van Gieson[1] noticed the presence of two anomalous bundles of white fibres which lay mesial of the hypoglossus nucleus. They were regarded as stray bundles from the posterior longitudinal fasciculus, or as bundles of association fibres for the cells of the hypoglossal nuclei.

CASE VI.—The case of Henle. It is mentioned above, and does not require further consideration.

To these cases I wish to add the following: Case I. is from a medulla cut in series by me in the laboratory of Prof. Obersteiner, in Vienna; Case II., from one cut by Dr. Kornfeld. For permission to publish this second case and for much assistance I am indebted to Prof. Obersteiner.

FIG. 2.



Section from the level of the sensory decussation. (*DLm.*)

Py, Pyramids. *Sgl*, Substantia gelatinosa. *Va*, Ascending root of trigeminus *Nc* and *Ng*, Nuclei of funiculus cuneatus and gracilis. Bundle (*a*) is plainly visible.

CASE I.—The antecedents of the medulla are unknown. As, however, it had been sent to the laboratory from the pathological department for use as a normal specimen, it may be assumed with comparative certainty that the patient had not died of a disease of the nervous system.

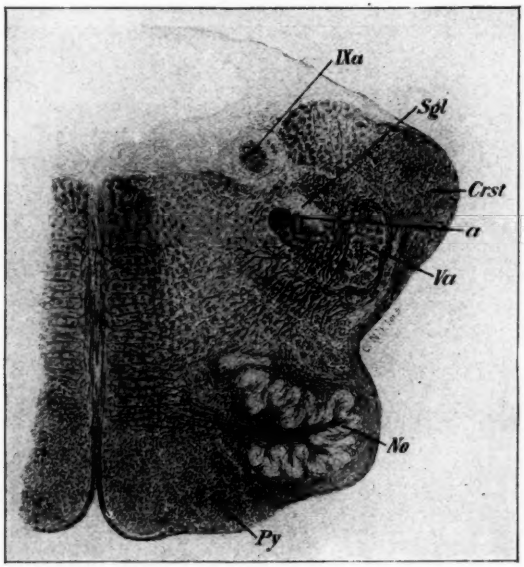
In the subsequent microscopical examination of the serial sections no pathological changes were demonstrable, but two anomalous bundles of nerve fibres were discovered.

The first of these, which I will term bundle *a*, takes origin at the

lower level of decussation of the pyramids. Here, as seen in Fig. 1, a number of fibres (*a*) separate from those engaged in the decussation (*DPy*), and proceed outward and slightly backward to a position anterior and internal to the head of the posterior horn (*Sgl*). They may be readily distinguished from the fibres of the funiculus lateralis (*fnl*) by reason of their greater calibre and consequent darker stain.

In Fig. 2, representing a section at the level of the sensory decussation (*DLm*), these fibres are seen to have united to form a circular, compact bundle, which occupies a position anterior and internal to the substantia gelatinosa (*Sgl*) and ascending root of the trigeminus (*Va*). It is surrounded by fine arching fibres, and has a diameter of about 0.50 mm. No connection between it and the cells of the nucleus funiculus cuneatus (*Nc*), or those of the nucleus funiculus gracilis (*Ng*) is demonstrable. (Compare Schaffer's case.)

FIG. 3.



Section through the upper third of the olive.

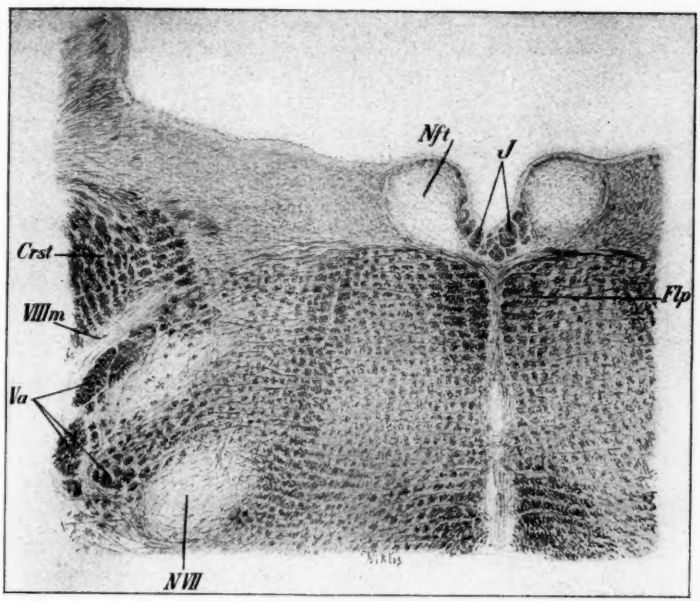
Py, Pyramid. *Crst*, Corpus restiforme. *Sgl*, Substantia gelatinosa. The abnormal bundle, *a*, lies to the mesial side of the ascending root of the trigeminus, *Va*, and is shown to be entirely independent of the ascending root of the glosso-pharyngeal, *IXa*.

In Fig. 3, representing a section of the upper third of the olive, the bundle (*a*) is seen to still occupy its position in proximity to the substantia gelatinosa, and close to the inferior border of the ascending root of the trigeminus. The bundle is somewhat smaller than at lower levels, owing possibly to a more compact arrangement of its fibres, but in general characteristics it is unchanged. The ascending root of the

glosso-pharyngeal (*IXa*), or respiratory bundle of Kraus, may be seen occupying a position dorsal and somewhat internal to the anomalous fasciculus. The entire independence of these structures is, however, well demonstrated. (Compare case of Henle.) No connection between the anomalous bundle and the corpus restiforme (*Crst*) is demonstrable upon this or upon other preparations.

At a somewhat higher level the bundle disappears, its fibres having separated and become lost among surrounding structures. The final destination of these fibres could not be determined, but there is no ground for assuming that they entered the corpus restiforme.

FIG. 4.

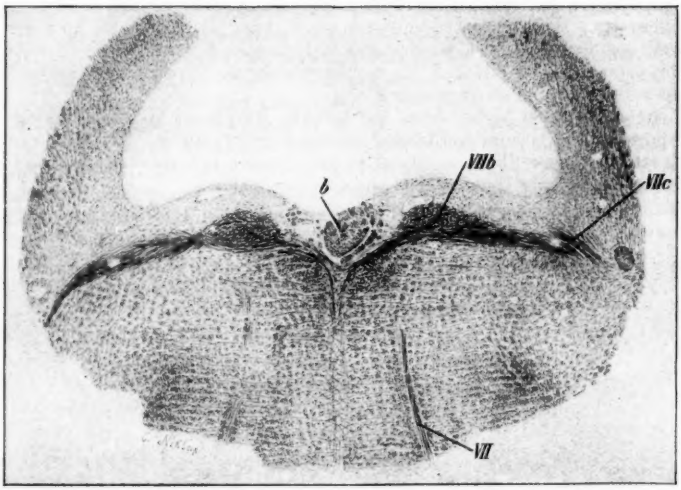


Section a little above the lower border of the pons.

N VII, Nucleus of the facial nerve. *VIIIm*, Median root of the auditory. *Crst*, Corpus restiforme. *Va*, Ascending root of the trigeminus. Bundle *a* is no longer to be seen. The second abnormal bundle, *J*, takes origin from the greatly enlarged nuclei funiculi teretes (*Nft*). *Flp*, Fasciculus longitudinalis posterior.

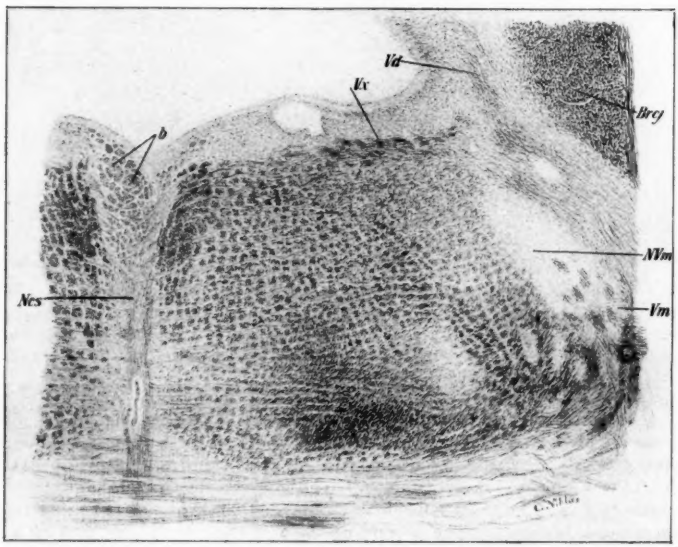
A second anomalous bundle appears in this medulla when we reach the level of exit of the eighth nerve. It is shown in Fig. 4 as a collection of small fasciculi (*J*) which run in a longitudinal direction near the floor of the fourth ventricle. To either side lie the nuclei funiculi teretis (*Nft*). These are enormously enlarged, and give off fibres to the anomalous fasciculi. The ascending root of the trigeminus is seen at *Va*, internal to the median root of the auditory (*VIIIm*). The structure at *N VII* is the facial nucleus. Bundle *a* has disappeared.

FIG. 5.



Bend of the knee of the facial (*VIIb* and *VIIc*). *VII*, Abducens nerve. The abnormal bundle, *b*, in the middle line.

FIG. 6.



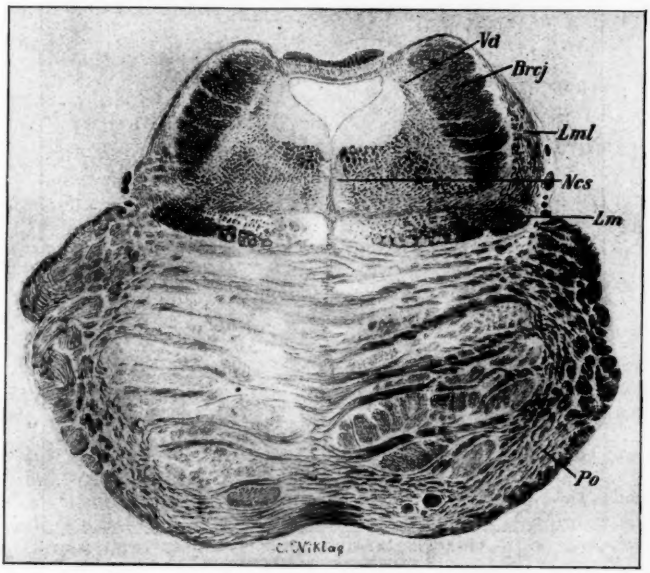
Region of the nucleus of the trigeminus.

NVm, Motor nucleus of trigeminus. *Vm*, Motor root of trigeminus. *Vx*, Crossed root. *Vd*, Ascending root. *Ncs*, Nucleus centralis superior. *b*, The anomalous bundle, which has subdivided into numerous smaller fasciculi.

At the level of the nucleus of the sixth nerve (Fig. 5) the greater number of the anomalous fasciculi have united to form a single large bundle (*b*). This is elliptical on cross section, and lies near the floor of the fourth ventricle, almost in the median line. No communication between the anomalous bundle and surrounding structures is demonstrable at this height. The structure at *VIIb* is the ascending arm of the root of the facial nerve.

Fig. 6 is taken from a section at the level of origin of the trigeminus. The motor nucleus and the motor root of this nerve lie at *Vm* and *Nvm* respectively, while its sensory nucleus and root lie external to these structures and internal to the brachium conjunctivum (*Brcj*). Its descending root is at *Vd*, and its crossed root at *Vx*. The anomalous structure *b* has subdivided into numerous smaller bundles which lie wedged in between the posterior longitudinal fasciculi. From the anomalous bundles fibres are given off which may be seen running toward the nucleus centralis superior (*Ncs*).

FIG. 7.



Section just below the corpora quadrigemina.

Vd, Descending root of trigeminus. *Brcj*, Brachium conjunctivum. *Lml*, Lateral. *Lm*, Mesial fillet. *Po*, Pons. *Ncs*, Nucleus centralis superior.

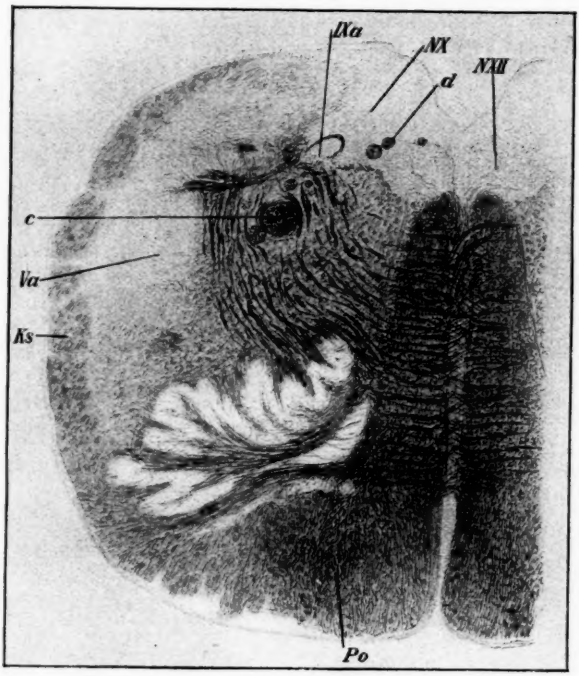
In Fig. 7, representing a section through the pons at the level of exit of the trigeminus, the nucleus centralis superior is seen to be of unusual size. The anomalous bundle has disappeared.

CASE II.—The sections are from a medulla showing marked pathological changes, consisting chiefly in multiple degenerations of nerve

centres and nerve roots in consequence of tabes dorsalis. In its pathological relations this case will be considered by Dr. Kornfeld in a later article. Of especial interest to us is the presence in this, as in Case I., of true heterotopia of white matter, which again manifests itself in the form of multiple unilateral bundles of nerve fibres.

The more important anomalous bundle, bundle *c*, may be first seen at the level of decussation of the pyramids, whence it passes upward in the lateral column, occupying a position to the mesial side of the substantia gelatinosa. It is circumscribed by arching fibres, and has a diameter at its point of greatest development of about 0.75 mm.

FIG. 8.



Section at calamus scriptorius.

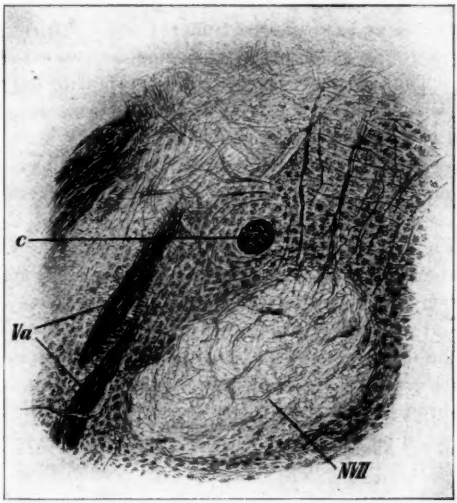
c and *d*, The abnormal bundles. *NXII*, Nucleus of hypoglossal nerve. *NX*, Nucleus of vagus. *Ks*, Lateral cerebellar tract. *Va*, The degenerated ascending root of the trigeminus. *IXa*, Degenerated ascending root of glosso-pharyngeal.

In Fig. 8, representing a cross section of the medulla at the lower third of the olive, the anomalous bundle is seen at *c*, to the mesial side of the (degenerated) ascending root of the trigeminus (*Va*), and midway between this structure and the respiratory fasciculus (*IXa*). The respiratory fasciculus is made up in this case of degenerated fibres, and it has consequently remained unstained (Pal method). A small sec-

ondary bundle is seen external and somewhat ventral of the bundle *c* (*N XII*). Upon the same side of the medulla, midway between the hypoglossal and the pneumogastric (*N X*) nuclei, run several small anomalous bundles of white fibres (*d*). These are of limited vertical extent, becoming lost laterally at the level of the upper third of the olive.

In Fig. 9, representing a section somewhat above the level of exit of the facial nerve, the abnormal bundle *c* is seen lying midway between the nucleus of the facial (*N VII*) and its emerging root (*Va*). It

FIG. 9.



Portion of a section from the lower part of pons.

N VII, Nucleus of facial nerve. *Va*, Emerging root of facial. The abnormal bundle, *c*, between the two.

is still nearly circular on cross section and remains sharply circumscribed. The secondary bundle has disappeared.

This bundle *c* may be traced to a point high up in the pons, where it finally becomes lost, due probably to a separation of its fibres at different levels, these then becoming indistinguishable from surrounding structures.*

CONCLUSIONS.—Bundles *a* and *c* correspond, in their location near the substantia gelatinosa, and in their general characteristics, with the bundle of Pick. The origin of bundle *a* from the fibres of the lateral column must be regarded as demonstrated. Although the origin of bundle *c* could not be definitely determined, it seems probable from analogy and

* The preparations were all stained according to Pal's modification of Weigert's hæmatoxylin method, and in part double stained by the subsequent use of alum-cochineal.

from the fact that it was first demonstrable at the level of crossing of the pyramids, that its fibres were derived from the same source. The superior termination could not be determined in the case of either bundle. It seems improbable, however, that either ended in the corpus restiforme as in Pick's case. I am much more inclined to the belief that they accompanied the fibres of the tegmentum and reached a much higher point. This seems especially probable in the case of bundle *c*, which could be traced as such to a point considerably above the level of exit of the facial nerve. An explanation as to the physiological significance of either bundle cannot be offered.

Bundle *b* excites especial interest from the fact of its running for the most part of its course as an unpaired fasciculus situated nearly in the median line. The fact that fibres may be seen entering it from the unusually developed nuclei funiculi teretes, and that at a higher level fibres leave it to stream in the direction of the greatly enlarged nucleus centralis superioris, suggests the possibility that it may serve as an abnormal communication between the cells of these structures.

A second, less plausible, explanation would be that the abnormal bundle was a misplaced conductor sonorus (*Klangstab*). This structure may be unilateral, and has been observed running along the floor of the fourth ventricle nearly parallel with its axis, in which case it has always been of unusual size.[8] The fibres of the conductor sonorus usually end, however, in the vicinity of the cells of the locus cæruleus and not in that of the superior central nucleus, and the explanation would not account for the extraordinary development of the nuclei funiculi teretes, which in this case could be seen actually giving off fibres to the anomalous bundle; hence it may, I think, be rejected as unsatisfactory.

The origin and the termination of the small bundles (*d*) seen running along the floor of the fourth ventricle between the pneumogastric and hypoglossal nuclei could not be determined.

Finally, from a study of our cases we may conclude that the central nervous system possesses, like other organs, a tendency toward occasional misplacement of its elements. This misplacement has, so far, never been observed to occur bilaterally. The most frequent and fairly constant histological anomaly appears to be the presence of a column of white nerve fibres (column *a* and *e*) arising at the level of decussation of the pyramids, and thence proceeding upward, occupying a position internal to the substantia gelatinosa. The fibres of this column may take origin from the lateral or from the posterior column. In certain instances these fibres have been traced above into the corpus restiforme, but this termination has seemed improbable in other cases in which the fibres have apparently extended to a much higher point.

NOTE.—Since writing the above article a communication has been received from Prof. Obersteiner, in which he announces that bundles *a*

and *c* have again been observed in his laboratory. They occurred unilaterally in separate medullæ, in each of which there was marked increase in size of the nuclei funiculi graciles. The *Revue Neurologique*, No. 464, contained a short reference to the fact that an abnormal bundle, corresponding with bundle *a*, has been repeatedly observed by Becterew and his pupil Riklinski.

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THE ISOLATED TYPE OF FRIEDREICH'S DISEASE.

BY H. J. MACKAY, M.B., C.M., EDIN.,
OF DEVIZES, ENGLAND.

DR. MACKENZIE'S interesting case recorded in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES for April reminds me that I have a very similar case under my care at the present time, of which the following notes may prove acceptable:

Mabel B., aged thirteen years, first seen in November, 1893. Had then tenderness in knees and ankles, with slight pyrexia. Two days later had precordial pain, and a soft mitral systolic bruit appeared. For this, which remained after the joint symptoms had cleared up, she was detained for some weeks in bed. Toward the end of her stay in bed some jerkiness of the facial muscles, of the head, and of the arm was noticed, which, taken with the rheumatic attack, aroused a suspicion of chorea. The twitchings, however, differed from those of chorea in being shorter in time and less extensive in range than choreic movements are generally. On first getting out of bed, about the middle of December, incoördination of gait was noticed, and ataxia was diagnosticated. She has one brother ten years of age, and two sisters—one fourteen and one three. All are healthy and have good knee-jerks. The mother is a healthy woman; her child-bearing history does not suggest syphilis. The mother's family are said to be all healthy, but one cousin of the patient died from "rheumatism, heart disease, and chorea." The father denies syphilis and exhibits no signs of it, and the family contribute no history of neurosis. The patient has always been a delicate child; had

jaundice at six years, but no illness thereafter until the rheumatic attack mentioned above.

On admission into the Devizes Cottage Hospital in February, 1894, the following was her condition: A pale, thin child, with slightly anxious expression, but cheerful, and no wise emotional. She has never menstruated. She lies in bed with her legs extended, the toes pointed, and the dorsa very prominent—the position resembling talipes equinus. This is equally marked when the pressure of the bedclothes is removed by using a cradle. There is a slight frown of the corrugators which is persistent, and occasional twitchings of the angles of the mouth, of the lips, and of the alæ of the nose. There is occasional slight tremor of the head, and jerking of the arms in the direction of the unilateral shrug of the shoulders, or slight extension of the forearm. On making her follow the finger sharply to right or left with her eyes, there is a slight but distinct lateral nystagmus. There is no lateral curvature, but the normal antero-posterior dorso-lumbar curve appears to be exaggerated.

Sensation is normal everywhere to touch, pain, and temperature, except for a patch of impaired acuteness on the outside of the right knee. There have been no "subjective" pains at any time; the special senses are normal, and so is the muscular sense. Voluntary power is good in the legs, as shown in resistance to passive movements. Nutrition of muscles is good, and their tenacity is normal. The plantar reflexes are absent; the abdominal and epigastric are present. There is no knee-jerk nor ankle clonus. Coördination is markedly impaired. She walks with the head carried well forward and the legs widely separated. The gait is ataxic, but not with the high step of locomotor ataxia. She has great difficulty in turning, and staggers as if she would fall in so doing. Romberg's sign present.

Electrical irritability to both currents is normal. The arms appear perfectly normal, but when the hands are quiet they frequently fall into a position of metacarpo-phalangeal extension, with some inter-phalangeal flexion. Her handwriting when compared with that of a year ago shows fine tremors and incoördination, which is absent from the specimens of earlier date.

Speech is not markedly affected, but she does not read so well as she did. The pupils are equal, and react to light and accommodation. The optic disks are normal, and there is no limitation of the visual fields, nor ocular palsy. She complains of giddiness occasionally. She has a systolic mitral murmur.

This may be a convenient place to record the following unpublished examples of the family type of the disease, two of the cases being under my care at present.

Case I.—Ellen W., aged twenty-one years, unmarried, was admitted into Devizes Cottage Hospital on March 24, 1891, for weakness of the lower limbs. This had been coming on since her eighth year. She remembers that at the first onset she used to stagger in walking, and as the weakness increased she would sometimes fall to the ground. For the last three or four years she has been able to get about the house only by supporting herself by the table or chairs. She complains, too, that during the last twelve months her hands have become more clumsy than they used to be. She is the youngest of a family of eight.

Four brothers are quite healthy. One sister died of "paralysis" (see Ann W., *infra*) at the age of nineteen. Another brother now suffers from paralysis (see Henry W., *infra*). The father is a healthy man, with no appearance of syphilis or alcoholic excess, and is sixty-two years of age. The mother, aged sixty-five years, is blind from double cataract, and for the last two years has been bedridden from rheumatoid arthritis (see genealogical table, *infra*). Nothing in the patient's habits or surroundings has any bearing on her case, but she remembers having had measles when seven years of age. Present condition: an anæmic girl of middle height, well nourished, with a cheerful but vacant expression.

She is easily moved to laughter of a silly and uncontrollable kind. Gets out of bed with difficulty, and cannot stand erect without assistance. When so assisted stands with legs wide apart, and the soles slightly inverted, as in talipes varus. On trying to walk there is extreme incoördination in her efforts.

The feet are drawn forward, raised with difficulty high from the ground, and planted anywhere with a jerk which threatens to throw her down. The toes are over-extended in the effort to stand, and the tendons stand out prominently on the dorsa of the feet. There is a tendency in the hands, when at rest, to extension at the metacarpo-phalangeal, and flexion at the inter-phalangeal joints.

Motor power fairly good in the legs, and apparently unaffected in the arms, as also is the irritability to both currents.

When lying on her back there is talipes equino-varus of both feet, with a shortened foot and over-arched instep. No rigidity on passive movements. Plantar reflex excessive on both sides. No ankle clonus.

No knee-jerk. Gluteal, abdominal, and scapular reflexes present. Pupils equal and active to light and accommodation. Bladder-control normal, but slight impairment of the power of the sphincter of the bowel.

Coördination extremely defective in legs and arms; muscular sense also impaired.

Has never had any pain, numbness, or tingling in her limbs. Cutaneous sensibility everywhere normal to touch, pain, and temperature. Her special senses are all acute. Her optic disks are natural. No trophic signs, but considerable lividity, with coldness of the feet and toes.

There is a well-marked lateral curvature, with convexity to the left. She has frequent twitching of the angles of the mouth when she articulates, which is more marked when she is excited. The attempt to depress the angles of the mouth and lower lips exaggerates this twitching into a clonic spasm of the depressor anguli oris. There is frequent bilateral, conjugate, horizontal nystagmus. No choreiform movements of limbs.

Speech nasal and "clipped," the words brought out with a jerk, and accompanied by a facial contortion.

Her memory is good, and her mental state clear but emotional. Menstruation deficient and bowels sluggish. Other symptoms normal, but she suffers much from palpitation and giddiness.

Case II.—Henry Thomas W., unmarried, aged twenty-six years, is the brother of the preceding. He was a healthy child till his sixth year, when he had an attack of measles, and, soon after, of scarlet fever.

Within a month after convalescence from the latter he complained of weakness in the legs and began to stagger. About his twelfth year the mother remarked that he used to go about stooping, with his head in front of him. After the twelfth year the lower limbs became helpless, two years later the hands and arms became weak, and since his twenty-second year he has been helpless in them, too. He is now unable to move, except to roll over in bed, and is carried about by his father.

Present condition: Sits in a chair with arms flexed and resting in his lap, and chin depressed on the sternum. His expression would be cheerful but for the constant grimaces produced by the facial muscular contractions, *e. g.*, the occipito-frontalis, each half of which contracts separately, orbiculares oris et oculi, levatores anguli oris, and zygomatici.

The hands are generally, but not immovably, hyperextended at the metacarpo-phalangeal, and flexed at the inter-phalangeal joints. The spine shows a well-marked postero-lateral curvature, with the convexity to the left, and there is a compensatory rotation of the pelvis. The trunk is bent forward, and the recti abdominis stand out as prominent ridges in the middle line anteriorly.

The thighs are flexed on the pelvis, and the legs on the thighs, extension at these joints being impossible. The knees are adducted and in contact, the left overlapping the right. From the approximated knees the flexed legs diverge to either side, so that the feet are widely apart. These latter are in a condition of extreme talipes equino-varus.

All the limbs are much shrunk in size, but what muscle remains is firm. There is no R. D. present, but the faradic and galvanic irritability are both diminished.

The pupil reacts to light and accommodation, and the control over bladder and bowel is unimpaired, but all other reflexes, cutaneous and deep, are abolished.

The speech is markedly affected, the effort to articulate bringing on spasmodic jerkings in nearly all the facial muscles. With these jerkings, and also apart from them, there is frequent oscillation of the eyeballs, but this is slower in time and wider in range than are usually seen in nystagmus. These oscillations are either bilateral (conjugate), or unilateral, and, generally horizontal, are sometimes oblique in direction, but never vertical.

Coördination cannot be tested on account of the crippled movement in the limbs. Sensation is everywhere acute. There have never been lightning pains, nor any dysæsthesiæ. The special senses are unaffected. The disks are normal. Ocular movements are much interfered with by incoördination of the muscles, which sometimes prevents him from fixing his line of vision on any object looked at, strabismus and diplopia coming on in the effort; but though jerky and incoördinate, the movements are normal in range.

His memory is good, and his mental capacity above the average. Slight exertion brings on extreme palpitation, and he has persistent vertigo, which, however, never goes on to nausea or vomiting. Otherwise his general health is good.

Case III.—Ann W., sister of the preceding, died at the age of nineteen years, and was never seen by me.

Her mother's account is that she was a healthy child at birth, and continued so till her fourth year, when she had measles. Very soon

after recovering from this complaint she became weakly and began to stagger in her walk. Three years later she developed a festinating gait. She could move about, but with difficulty and frequent falls, till her tenth year, after which she had to be carried. At this date she could sew and use her arms, but six years later—in her sixteenth year—the upper arms began to get weak, and this increased steadily till her death. Before her death she appears to have been absolutely helpless as regards movements of arms, legs, head, or trunk. She became unable to speak three days before death, but was able to swallow to the last.

She always had control of the bladder and bowel, never complained of pain, had the use of all her senses, and her mind remained clear throughout.

Her feet and hands in the later stages had the same deformity that her brother's now have.

REMARKS.—The isolated case is of interest—if time should confirm the diagnosis arrived at—on account of the quick progress of the disease and the early stage at which the diagnosis was possible. This had to be made between locomotor ataxia, cerebellar tumor, and chorea. The exclusion of the two former was possible on the line adopted by Dr. Mackenzie; that of chorea was more difficult.

The similarity of the early stages of Friedreich to chorea, which has already misled such observers as Ladame, Moxon, Ormerod, and others, was here increased by the antecedent rheumatism and the presence of a systolic murmur.

The facts which led me, in the face of such odds, to conclude in favor of ataxia were the following:

1. *The distribution.* The hands were unaffected except to the slight extent discernible in writing; the arms were only involved to the extent of a slight muscular twitch; the legs, which showed no twitching whatever, were the seat of extreme incoördination.

It is usual in chorea to have both legs the seat of incoördination, while the hands show neither involuntary movements nor weakness.

2. *The nature of the twitchings.* (*a*) These could not well be described as clonic spasms, if the latter be taken to mean increased as well as involuntary muscular action. They were more of the nature of muscular tremors, being slighter in range and slower in time than choreic spasms. (*b*) They affected either single muscles (levator anguli oris), or a part of a muscle (anterior portion of trapezius or part of triceps); never co-ordinated groups of muscles (supinators, adductors, or rotators), as generally happens in chorea. (*c*) They were not increased by emotion or by voluntary effort in this case.

(In the cases of Henry and Ellen W. the muscular twitchings were increased by effort or emotion, however; probably this occurs in the later stages of the disease.)

3. *The gait.* This was distinct from the shuffling, irregular gait of chorea. The head was kept well forward; the arms held apart from the

body, as if to balance; the feet straddled widely to obtain a larger base; the pace was quickened, as if to call momentum to the aid of equilibrium; the frequent stagger was not due to jerkiness of the muscles of the leg or the trunk throwing the body off its balance, as in chorea, but suggested the reel of vertigo or of intoxication.

4. *Static equilibrium.* I have never seen Romberg's sign typically present in chorea. A sudden muscular contraction may disturb the child's balance, but in the interval between the spasms equilibrium is maintained. In Friedreich's disease the sway which precedes a fall is evidently due to a persistent interference with the motor side of the co-ordinating mechanism, and not to a momentary disturbance of it, as in chorea.

5. *The nystagmus.* While unilateral and irregular spasm of the ocular muscles is met with in chorea, it is doubtful if nystagmus—bilateral, symmetrical, and rhythmical—ever occurs. I have never seen it recorded in connection with chorea.

6. *The state of the feet.* The gradual onset of talipes with incoordination in a child should always raise a suspicion of Friedreich's disease, pointing as it does to structural cord change. Dr. Bristowe, I am aware, mentions a case of chorea (Fagge's *Medicine*, vol. i. p. 734, footnote) in which there was a tendency to talipes equino-varus. If the description be read, however, it will be found to accurately describe the condition present in H. W.'s case, which is undoubtedly one of ataxia. With all deference to a great physician, one may be pardoned for doubting whether the same disease was not present in both cases.

As regards the family cases, one point of interest in connection with them is the sequence of the disease, in two cases immediate, to attacks of measles. A similar sequence occurred in Dr. Mackenzie's case. Acute fevers are, of course, recognized as etiological factors in the genesis of the disease, but I have not had time to consult the literature of the subject as to the preponderating influence of measles in this respect.

Remembering, however, the rôle of measles as a neurosis-producer,[1] one would not be surprised if the chronic cord change of Friedreich's disease frequently owned such an attack as a factor in its etiology.

The distortion of the hands and feet in all these cases points to this being a pretty constant feature of the disease. In the hand, in the later stages, the deformity resembles the claw-hand of progressive muscular

[1] The sequelæ reported include disseminated myelitis (Barlow, Lancet, ii. 1884), cerebro-spinal meningitis (Cheadle, *ibid.*), various palsies (Ormerod, *ibid.*, and Colcott Fox, *ibid.*, i. 1887), acute ascending paralysis (Negrie, *ibid.*, ii. 1887), peripheral neuritis (Monro, *ibid.*, i. 1894), and numerous others referred to by Monro, and recorded by Ross and Bury, and by Kop.

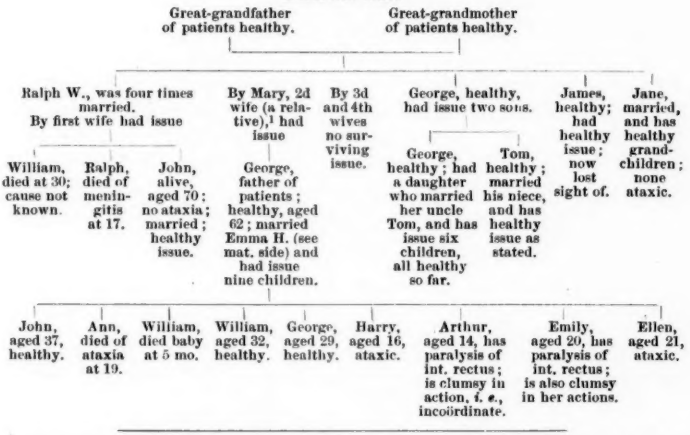
I have myself notes of four cases of non-tubercular meningitis following measles, without ear complications.

atrophy, or of ulnar lesion. In the foot the early stage resembles that of paralysis of the dorsi-flexors. The later stages appear to be generally a condition of extreme talipes equino-varus. The extreme and almost absolute fixation of head, trunk, and lower limbs, in the second of the family cases (Henry W.), by the preponderant action of the flexors throughout the body, produced a clinical picture of distortion following paralysis, such as one seldom sees surpassed in nervous disease.

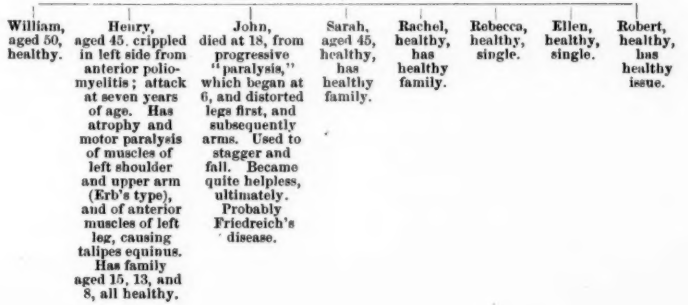
The genealogical table of this family suggests several matters of interest. One is the consanguinity existing between the grandparents of the patients, a degree of consanguinity, however, which could hardly have been greater than that between the uncle and niece in a collateral branch of the family.

GENEALOGICAL CHART OF THE W. FAMILY.

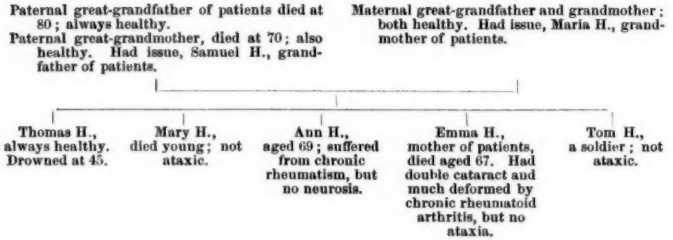
Paternal side.


Great-grandfather of patients healthy.
Great-grandmother of patients healthy.
Ralph W., was four times married. By first wife had issue
By Mary, 2d wife (a relative),[1] had issue
By 3d and 4th wives no surviving issue.
George, healthy, had issue two sons.
James, healthy; had healthy issue; now lost sight of.
Jane, married, and has healthy grandchildren; none ataxic.
William, died at 30; cause not known.
Ralph, died of meningitis at 17.
John, alive, aged 70; no ataxia; married; healthy issue.
George, father of patients; healthy, aged 62; married Emma H. (see mat. side) and had issue nine children.
George, healthy; had a daughter who married her uncle Tom, and has issue six children, all healthy so far.
Tom, healthy; married his niece, and has healthy issue as stated.
John, aged 37, healthy.
Ann, died of ataxia at 19.
William, died baby at 5 mo.
William, aged 32, healthy.
George, aged 29, healthy.
Harry, aged 16, ataxic.
Arthur, aged 14, has paralysis of int. rectus; is clumsy in action, i. e., incoördinate.
Emily, aged 20, has paralysis of int. rectus; is also clumsy in her actions.
Ellen, aged 21, ataxic.

[1] The antecedents of this second wife, though of great interest, cannot be unearthed. It is certain that she was a relative of her husband, Ralph, but the degree of kinship cannot be ascertained. She had one sister and two brothers—all healthy. The sister, name unknown, had a son named Robert W., before her marriage. She subsequently married and had no further issue. The son, Robert W. (a cousin of George W., the father of the recorded patients), married a healthy woman, and had issue as under, the children being second cousins of the patients above.


William, aged 50, healthy.
Henry, aged 45, crippled in left side from anterior poliomyelitis; attack at seven years of age. Has atrophy and motor paralysis of muscles of left shoulder and upper arm (Erb's type), and of anterior muscles of left leg, causing talipes equinus. Has family aged 15, 13, and 8, all healthy.
John, died at 18, from progressive "paralysis," which began at 6, and distorted legs first, and subsequently arms. Used to stagger and fall. Became quite helpless, ultimately. Probably Friedreich's disease.
Sarah, aged 45, healthy, has healthy family.
Rachel, healthy, has healthy family.
Rebecca, healthy, single.
Ellen, healthy, single.
Robert, healthy, has healthy issue.

Maternal side.


Paternal great-grandfather of patients died at 80; always healthy.
Paternal great-grandmother, died at 70; also healthy. Had issue, Samuel H., grandfather of patients.
Maternal great-grandfather and grandmother; both healthy. Had issue, Maria H., grandmother of patients.
Thomas H., always healthy. Drowned at 45.
Mary H., died young; not ataxic.
Ann H., aged 69; suffered from chronic rheumatism, but no neurosis.
Emma H., mother of patients, died aged 67. Had double cataract and much deformed by chronic rheumatoid arthritis, but no ataxia.
Tom H., a soldier; not ataxic.

In the latter case no ataxia has so far developed in the issue of the marriage. The disease, however, reappears in the descendants of the second wife's sister, and this appears to point to the family to which the second wife and her sister belonged as being the source of the taint. Thus, of seventeen second cousins—the descendants of these two sisters—hereditary ataxia appears in four, ocular and other neuroses in two, and acute atrophic paralysis in one. Rutimeyer has recorded (*Virchow's Archiv*, 1883) a similar association of anterior poliomyelitis with Friedreich's disease, occurring in a branch of the Blattner family reported by him.

NOTES ON THE OBSERVATION OF MALARIAL ORGANISMS IN CONNECTION WITH ENTERIC FEVER.[1]

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The possibility of typhoid and malarial fever manifesting their symptoms at one time in the same individual has afforded a fertile subject for debate for many years, with the result that the belief in the simultaneous action of the two infections has been gradually losing advocates. The theory originally advanced by Woodward, that a "typho-malarial fever" exists as a specific and independent disease, other than a typhoid fever made irregular by admixture with malarial poison, has been more and more abandoned by competent clinicians, as it subsequently was by Woodward himself, although it is occasionally revived in the South and Southwest in discussions upon the continued fevers of the South; especially in newly settled localities whose success as health resorts is closely connected with a belief in the entire absence of typhoid fever.

An exceedingly interesting paper was read before this Association six

[1] A paper read before the Association of American Physicians at the American Medical Congress at Washington, on May 30, 1894.

years ago by Dr. Johnston,[1] in which he summarized data in regard to the relation of typhoid fever to fevers of malarial origin, based upon replies to questions sent to 350 physicians residing along our Atlantic and Gulf seaboards. Dr. Johnston's contribution to this subject is familiar to us all, but I desire to refer briefly to one or two of its features. The research elucidated very strikingly the variations in type of enteric fever depending upon locality.

Two of the questions propounded were the following:

1. "Do you recognize in your practice a distinct type of continued fever which is neither malarial nor typhoid, or one which is a compound of both—a typho-malarial fever?"

2. "Do you think that the typhoid fever which you see is modified by malarial infection?"

The answers to the first question conform to geographical distribution, the existence of a "typho-malarial fever" being very generally denied in the Northeastern States, and affirmed by rather more than one-half the answers received from eastern North Carolina, South Carolina, Georgia, and Florida.

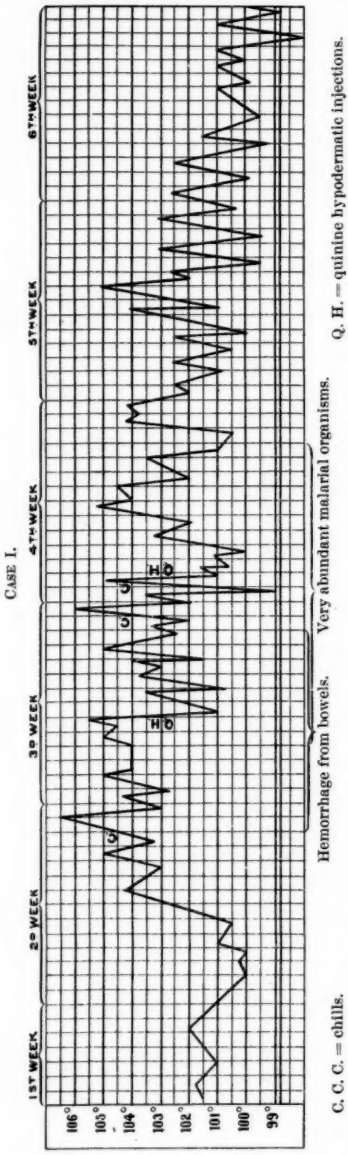
In answer to the second question, some stated their belief that in certain localities in the South "all the forms of fever seem to be more or less complicated with malaria, so much so that it is almost impossible to treat it successfully without the use of quinine in considerable quantities." Others living further to the northeast affirm that "there are no mixed continued fevers."

The belief is, I think, almost universal that typhoid fever, as observed in New York, is uncomplicated by malarial infection, and that quinine, except as a tonic in convalescence, is worse than useless in its treatment. For this reason the following cases, seen in New York last summer and autumn, are of unusual interest.

Case I.—The patient, J. McN., male, a native of the United States, forty-three years of age, was admitted to the Presbyterian Hospital, New York, on August 18, 1893. He denied having had ague, and gave no previous history of interest excepting that for the past twelve years he had indulged at intervals in the opium habit, taking of late as much as twenty grains per diem. For three days he had been ill with headache, vertigo, prostration, epistaxis, and slight cough, with mucous expectoration. On admission, his appearance suggested typhoid fever, and he was treated accordingly by the Brand method of cold tub-bathing. There were slight enlargement of the splenic area of dulness, great prostration, diarrhœa, and a typical typhoid tongue—dry, coated on the dorsum, with thin red margins and swollen papillæ. There was continued fever which lasted for seven weeks, and during this period the patient developed the following symptoms: a genuine typhoid eruption, there being some forty distinct rose spots on the abdomen and chest, which appeared in

[1] "On the Geographical Distribution of Typhoid Fever in the United States; its Relation to Fevers of Malarial Origin," etc. W. W. Johnston: Trans. Assoc. Amer. Phys., 1888, vol. iii. p. 8.

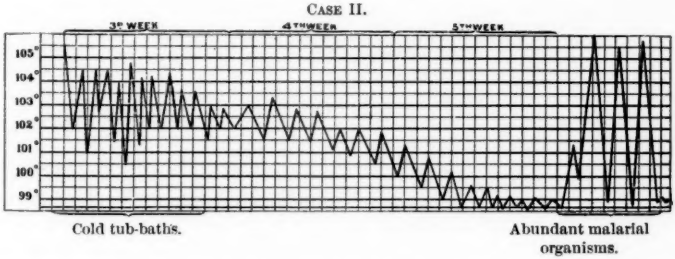
successive crops; hemorrhages from the bowels, of which there were four or five of considerable amount, tympanites, bronchial catarrh, slight albuminuria with granular casts, semi-stupor and delirium, subsultus, great prostration and emaciation, and the facies of the typhoid condition. On the thirteenth day of the illness there was a severe chill, lasting about three-quarters of an hour, and so violent that the patient shook the bed. It was accompanied by a rise of temperature to 106.6° F., but there was no sweating. During the third week two other chills occurred of equal violence. As the first chill took place before the hemorrhage and also before the eruption became decisive, and as there was nowhere evidence of suppuration, it appeared possible that there might be an error in the diagnosis, and the blood was carefully examined for the malarial plasmodium. It was found in exceptionally large numbers invading the red blood-corpuscles and also independent of them. When the next chill and exacerbation of temperature occurred quinine was given hypodermatically with the effect of reducing the temperature 4.5° (105.5° to 101° F.). This treatment was several times repeated, and on one occasion, at the end of the third week, the temperature was temporarily reduced from 106.4° to 99° F. No more chills occurred after the beginning of the fourth week, but the use of quinine was continued by the mouth, and the bathing, previously interrupted by the hemorrhages, was resumed. The patient made a good recovery, and after fifty-five days in the hospital was discharged, cured.



I regret that no examination of the stools for typhoid bacilli was made, but in view of the distinct eruption, epistaxis, intestinal hemorrhages, and tympanites, in con-

nection with all the other symptoms, there can be no reasonable doubt that typhoid fever was present, and repeated examinations of the blood proved the presence of the malarial organisms on each occasion. (There was nothing in the character of the stools to indicate dysentery.) I have never seen a more complete picture of enteric fever as regards all the symptoms, excepting the chills and irregular temperature curve.

CASE II.—The patient, J. J., an Irish laborer, thirty years old, was admitted to the New York Hospital on July 25, 1893. Two weeks before admission to the hospital he had had five or six chills of considerable severity, accompanied by nausea, vomiting, slight diarrhœa, and fever. He had complained also of severe frontal headache. On admission, the spleen could be palpated half an inch below the free border of the ribs, and two or three dozen rose spots were found upon the chest, abdomen, and back. The Ehrlich reaction was present. There were tympanites and slight albuminuria with decided prostration. The tongue was dry, coated on the dorsum, with thin red margins and prominent papillæ.



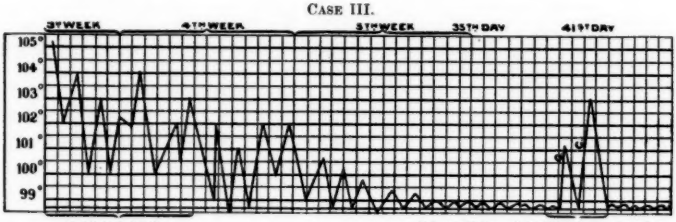
The patient was treated by cold tub-bathing without medicine. He received in all thirty baths. The fever ran as typical a typhoid course as one often finds when cold "tubbing" is employed; the maximum temperature was 104.5° F., and it became normal upon the thirty-first day. The Ehrlich reaction disappeared. The temperature remained below 99.4° F. for several days, and on the thirty-seventh day suddenly rose to 106° F., and was accompanied by profuse perspiration. There were chills and high fever (105.5° F.) on each of the two successive days at the same hour, and Dr. Ferguson, the pathologist of the hospital, who examined the blood for me, found malarial organisms in abundance in the blood. From the continued giving of quinine the patient had no more chills or fever, and in a few days he was discharged, cured.

There can be no question that this was a genuine case of typhoid fever. The course and duration of the fever, the headache, diarrhœa, enlarged spleen, the appearance of the tongue, the facies and hebetude of the patient, and the distinct rose spots all combined to make the case very typical. The following case is very similar to the preceding:

CASE III.—The patient, W. L., male, U. S., a laborer, twenty-seven years old, was received at the New York Hospital on the nineteenth day of his illness. His temperature then was 104.5° F.; respiration, 24;

pulse, 100. There was a distinct eruption of rose spots on the abdomen and thorax, a red, dry tongue with swollen papillæ at the tip, great prostration, and the facies of enteric fever. The spleen was palpated half an inch below the free border of the ribs. The patient was treated by cold tub-bathing, receiving twenty-one baths in all. He made a good recovery, and after ten days of normal temperature he was allowed the freedom of the ward. He was about to be discharged as cured on

CASE III.



Cold tub-baths.

C. C. = chills.

Pigment found in blood corpuscles, but no germs.

the forty-first day, when he had a severe chill, lasting half an hour, followed by fever and sweating. The chill was repeated the next afternoon at about the same time, also with increased temperature. Quinine was promptly given and the blood was not examined until the patient had received twenty grains. No malarial organisms were then found, but abundant malarial pigment granules were contained in the blood cells. Under the continued use of quinine, the patient recovered three days later.

In the first case reported the plasmodium became active during the height of the typhoid disease. In the second and third cases the malarial symptoms remained latent (although the plasmodium must have been already present) until the force of the enteric infection had been completely expended, when they assailed a body weakened by a fever of considerable duration. This is of interest in view of the well-known fact that in those who are subject to malarial paroxysms the malarial poison often takes advantage of lowered conditions of the system produced by fatigue, exposure, inanition, constipation, etc. It may be noted that neither of these patients recalled having had previous attacks of ague. With regard to the first case, it is to be observed that the typhoid fever, at first very mild, was immediately greatly aggravated by the malarial complication. The patient's condition was so reduced by it that his death seemed certain. The sudden reductions in temperature which took place bore a definite relation to the giving of quinine, and not to the hemorrhages. The chills also were not connected with the hemorrhages.

The advocates of typho-malarial fever as a distinct disease usually emphasize the absence of rose spots as invariable.[1] Others, who deny

[1] Squire: Epidemiol. Soc. of London. Lancet, January 8, 1887, p 73.

the possibility of the coexistence of malarial and enteric fever, account in various ways for the chills and sweating in cases presenting a similar clinical picture to those above reported, they not having found the malarial plasmodium in the blood.

Thus, Bouveret[1] reports four such cases in which he believed the modified symptoms were due to a slow and irregular elimination of toxic material, a large volume of which is sometimes thrown into the blood at once; and Fränkel[2] reports a similar case in which he thought the symptoms might be due to a thrombus in a mesenteric vessel, with suppuration and possibly miliary abscesses in the liver.

Professor Osler, in a recent number of the *Johns Hopkins Hospital Reports*, vol. iv. No. 1, mentions three interesting cases in which malarial fever was present with the plasmodium within a few months of the occurrence of enteric fever and once within a few days, but as the result of his experience he says: "There was no case with the characters of the two diseases so blended that it seemed a compound or hybrid malady, nor was there an instance in which the manifestations of the two diseases were concurrent."

It is not well to draw hasty conclusions from a very limited number of cases, but there is a kind of evidence which does not need to be multiplied to be convincing; and it seems to me, from the observations of the few cases herewith presented, that while it is unwise to accept the term typho-malarial fever as indicating a third form of disease, which is neither typhoid fever nor malarial fever, it cannot be denied that the two latter diseases may coexist.

Case I. certainly proves that they may do so, and this in a part of the country in which this form of double infection is most unlooked for, and the other cases, although perhaps less striking, are at least corroborative.

A STUDY OF ONE HUNDRED AND THIRTY-EIGHT CASES OF POTT'S PARALYSIS.

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The matter of this paper has been gleaned from all the printed sources available, and from an original study of one hundred and thirty-eight cases, one hundred and five of them being collected from the literature of the subject, the rest, thirty-three in number, being all the cases treated at the Boston Children's Hospital before 1891, of which a fairly full account could be obtained. For permission to use this last material I

[1] Lyon Médical, June 5, 12, 1892. [2] Lyon Médical, June 19, 1892.

am indebted to Drs. E. H. Bradford and H. L. Burrell, of the surgical staff of the hospital.

PATHOLOGY.—The work of Michaud,[1] Erb,[2] Courjon,[3] Elliot,[4] Gowers,[5] and others, has made the usual course of the process clear.

In the vast majority of cases the caseous osteitis of the bodies of the vertebræ extends back, involves first the posterior common ligament, which becomes ulcerated, and then the external surface of the dura, making a pachymeningitis externa. The tumor so formed projects into the vertebral canal, presses on the cord, sometimes forcing the cord against the laminæ, and thus sets up an interstitial myelitis of greater or less extent, from which ascending and descending degenerations may take their origin.

Macroscopically, the dura is seen to be thickened, now in one or two spots, again like a ring, again generally. Regularly, however, it is the outer surface which shows a change; the inner surface remains unaffected. The pia and arachnoid appear normal. The nerve roots are frequently found compressed by masses of exudation in the inter-vertebral foramina. The cord in early paralysis may show on gross examination absolutely no change in size, color or consistency, or it may look pale or red at the point of compression, and show some softening; later it may show hardening instead. The shape may be irregular, and the size may be reduced even to one-fifth of the normal calibre. Changes above and below the point of compression are not usually to be seen with the naked eye, except by the help of staining fluids.

Microscopically, one sees in the dura outside the normal inner layer a fibrous layer containing many leucocytes; the outer edge of this merges into a mass of caseous pus. At a late stage the dura may be seen to be made up of a mass of fibro-cartilaginous plaques. If compression has lasted some time the cord shows at the site of the lesion a considerable increase and thickening of interstitial connective tissue, a thickening and fatty degeneration of the intima of the vessels, and throughout the cross section many granular corpuscles, globules of myelin, and corpora amylacea; the gray matter may be distinguishable from the white only after staining, and then with difficulty; the axis cylinders are swollen or broken down; in the central gray matter the ganglion cells are swollen, vacuoles are formed, and there is here and there pigment deposited. As the seat of the caries is in the bodies of the vertebræ, and as the anterior portions of the cord are usually held close to the posterior surfaces of the vertebræ, while the posterior portion is five or six lines from that face of the canal, the meningitis is ordinarily anterior, and the myelitis first appears in that part of the cord. The myelitis may, however, be more or less unilateral, and may vary in extent from a slight infiltration to a complete destruction of the cord. There may be no changes except at the point of compression, or, above and below,

destructive processes may go on, and these, though they may affect the gray matter to a variable extent in either direction, are especially apt to cause alterations in the white matter. Above, degeneration[6] usually follows the posterior columns—the direct cerebellar tract when the lesion is above the lumbar enlargement, the ascending antero-lateral tract of Gowers, and high up the fasciculi graciles, even to the medulla, while below, degeneration goes on in the pyramidal tracts, direct and lateral, even to the cauda equina. In these consecutive lesions the microscope shows, in the gray matter, the same changes, though less advanced, that are seen at the point of compression, and in the white matter interstitial proliferation of connective tissue and degeneration of nerve fibres. This degeneration is not generally uniform on the two sides, and is, to a certain extent, variable in the path followed. Such is the customary pathological process, but there may also rarely be pressure paralysis in which the pressure is exerted by bone, as by an acute angle of bone, by a sudden dislocation of one vertebra on another, by an obtuse angle of bone over which the cord is tighly stretched, or by a sequestrum[7] detached within the canal; or, again, the pressure may be caused by an abscess[8 9] encroaching on the canal.

Moreover, the paralysis may be due, as Elliott[4] has pointed out, to an obstruction of the blood supply, in which case there may be no myelitis, only an acute œdema, and recovery may be very rapid.

Such obstruction is easy, as the vessels are small, do not anastomose freely, are exposed on the surface of the cord, and enter through foramina easily blocked by exudation.

Finally, it is possible, though most rarely does it happen, for inflammation to spread to the cord, either by contiguity through dura and pia or along the nerve roots, and there to set up a myelitis by extension.

In contrast to the class of cases showing paralysis without lesion of the cord, there are those cases which show compression and atrophy of the cord without paralysis,[10] and complementary to these last are the recoveries from paralysis; for in these, examination at times finds well-marked sclerosis existing, while the functional activity of the cord is not lessened. The closeness of the cord to the anterior wall of the canal, and the facts that the disease of the bone is usually in the bodies of the vertebræ and that the tracts conducting motor impulses are more superficial than those transmitting sensory impulses, are reasons why the paralysis is usually motor before it is sensory. In some cases, however, there is relatively great destruction of the cord in the posterior columns, due to pressure on the laminæ, and in these there may be recovery of motion and sensation but permanent ataxia.

Showing the proportion of the causes of paralysis, Poore[11] says that autopsies in 66 cases show a cord compressed 53 times by thickened meninges; 4 times by dislocation; once each by enlargement of odontoid,

by an acute angle of rapid formation, by dead bone, by a blunt angle over which the cord was stretched, by diseased bone which had given way, and three times by an abscess which had burst into the spinal canal.

Of the 35 deaths in the cases studied, records of autopsies have been found in only 22, and of these only 8 were complete, that is, included a microscopical study of the cord. In those examinations, made by various observers, there was reported a pachymeningitis in 5; signs of myelitis in 4; softening with hemorrhagic infiltration in 1; sclerosis in 3; secondary degeneration, ascending and descending, in 5, and neuritis in 2 cases.

In every case the cord was found affected, and in most cases the meninges.

In tabulating the 14 macroscopic examinations, it is found that an alteration of the shape or size of the cord, a flattening or a lessening, is the commonest lesion, occurring 8 times; softening is reported 6 times, while sclerosis was evident only once, a pachymeningitis was present in 5 cases, and it is clearly stated in two instances that the cord looked normal.

SYMPTOMATOLOGY.—(*a.*) This cannot be prefaced better than by the classical clinical history of Pott:[17]

> The patient "at first complains of being very soon tired, is languid, listless, and unwilling to move much or at all briskly; in no great length of time after this he may be observed frequently to trip and stumble, although there be no impediment in his way, and whenever he attempts to move briskly he finds that his legs involuntarily cross each other, by which he is frequently thrown down, and that without stumbling; upon endeavoring to stand still and erect without support, even for a few moments, his knees give way and bend forward; when the distemper is a little further advanced it will be found that he cannot, without much difficulty and deliberation, direct either of his feet precisely to any exact point, and very soon after this both thighs and legs lose a great deal of their natural sensibility, and become perfectly useless for all the purposes of locomotion.
>
> "Some are rendered totally and absolutely incapable not only of walking, but of using their legs in any manner, others can make shift to get about with the help of crutches, or by grasping their thighs just above the knees with both hands; some can sit in an armed chair without much trouble or fatigue, others cannot sit up with any help; some retain such a degree of power of using their legs, as to be able to shift their posture when in bed; others have no such power, and are obliged to be moved on all occasions.
>
> "The first return of the power of motion in the limbs is rather disagreeable, the motions being involuntary and of the spasmodic kind, principally in the night, and generally attended with a sense of pain in all the muscles concerned.
>
> "In the milder kind of case, the power of voluntary motion generally soon follows the involuntary.
>
> "The knees and ankles by degrees lose their stiffness, and the relaxation of the latter enables the patient to set his feet flat upon the ground; the certain mark that the power of walking will soon follow."

(*b.*) *Prodromal period.* There may or may not be a prodromal period characterized by sensory disturbances.

There may be pain, varying much in degree; it may be girdle-like, or

there may be persistent neuralgias at any spot, usually of abdomen or legs. Possible also are paræsthesiæ, as tinglings or sensations of heat or cold, hyperæsthesia and anæsthesia; this last is usually in spots. There are said to be sometimes herpetic or bullous eruptions over areas supplied by nerves from affected regions.[2]

(*c.*) *Paralysis of motion.* The paralysis which is characteristic of myelitis varies from a slight weakness of a few muscles, not to be noted without close observation, to a complete loss of power of most of the muscles of the body, so that the patient lies prostrate.

Though usually a paraplegia, the paralysis may involve all parts of the body supplied by spinal nerves, or it may be a monoplegia,[18] or a hemiplegia;[19] either, too, may develop after a time into a paraplegia; there may be a brachial paraplegia, and later this may extend and involve the legs.[20]

In severe cases the abdominal and respiratory muscles may be affected. The legs are very often affected unequally.*

Of 59 cases reported by Gibney,[21] 24 (40.6 per cent.) had complete paralysis, and of these 4 had also incomplete cervical paraplegia. Of 59 cases analyzed by Taylor and Lovett,[22] 32 (54.2 per cent.) had complete paralysis; 3 had paralysis of the arms. Of Myers'[23] 218 cases, paralysis affected the arms in 7. Of the 138 cases studied for this paper, 61 (44 per cent.) had complete paralysis, and 26 had more than a simple paraplegia. Of the 256 cases thus available for comparison, 117 (45 per cent.) had complete paralysis of the limbs affected.

In some cases the arms are first paralyzed, while the legs are free. This may be due to the fact that the lesion is at the level of the cervical enlargement, and in this place first attacks the anterior roots for upper extremities; in this form of palsy muscular atrophy and absence of reflex actions form a characteristic feature;[24,25] or if the lesion is in upper part of cervical cord, the motor paths in the antero-lateral columns which belong to the upper extremities may be attacked first; in this case reflex actions would be present. In a few cases, the compression being limited to one-half the cord has caused a hemiplegia with crossed anæsthesia, a typical unilateral lesion.[19]

(*d.*) *Paralysis of sensation.* "Some impairment of sensation is found in all Pott's paralyses, but it is usually so slight as only to be recognized by the most delicate tests, and for a short time."[26]

Very frequently, however, sensation in paralyzed parts is decidedly dulled, usually some time after the muscular weakness is noted, and there may be almost complete anæsthesia of all the body below the site of compression. This is significant of a very extensive transverse myelitis.

* There have been cases where paralysis of arms followed paralysis of legs, when the lesion was in the dorsal spine. This may be due to an ascending myelitis, which is not rare, or to ascending degeneration of the lateral columns, which is quite rare.

There may be, too, hemi-anæsthesia, or areas of anæsthesia; De Jonge[27] describes a case with increased sensibility in the paralyzed parts.

There may be, though usually there is not, pain of a constant, heavy, aching character all over the paralyzed limbs. There may be also paræsthesia and hyperæsthesia, all of these sensory symptoms being due to pressure or myelitis of the posterior columns.

Generally tactile sense disappears first, then the sense of temperature, and last the sense of pain. Michaud[1] quotes a case in which there was paralysis of sensation and not of motion, and there are several cases in which the sensation was found dull before muscular weakness was noted.

Taylor and Lovett[22] found out of 59 cases 15 (25.4 per cent.) with incomplete sensory paralysis.

Of the 138 cases, 71 (51.4 per cent.) had some notable impairment of sensation.

(*e.*) *Spasms and contractures.* In the prodromal period there may be twitchings of various muscles, due to irritation of the anterior nerve-roots; but generally at first the paralyzed muscles are completely relaxed, quiet, and soft; the joints also are relaxed and make no opposition to passive motion. In the later stages, with the advent of secondary degeneration in the cord, the flaccid muscles may become tense and rigid; usually at first the legs are extended stiffly and the feet are held in slight equino-varus; later a change comes on, slowly or suddenly, sometimes while the patient is asleep; the thighs become flexed on the pelvis, adducted, the legs flexed on the thighs, and the feet frequently take the position of calcaneo-valgus. If the limbs are extended again they frequently flex in a jerk without any apparent cause.

Sometimes there is present what is called "clasp knife" rigidity (for example, if, when the knee is flexed, the leg is gradually straightened, it moves smoothly and easily, till full extension is nearly reached, and then it suddenly becomes rigidly extended, and a good deal of force is required to bend the knee again).

At first abnormal positions can be readily passively corrected, but later contractions set in, and the legs become fixed in the position adopted.

Of the 138 cases 19 are noted as having rigid muscles.

(*f.*) *Bladder and rectum.* In severe cases either the bladder or the rectum, or both, may be affected; usually at first there is some difficulty in micturition—slowness in starting the stream—but later there is true incontinence.

The anal sphincter may be paralyzed and the contents of the bladder and rectum may be involuntarily evacuated consciously or unconsciously. Incontinence may result some months after symptoms of complete paraplegia have existed, apparently from downward extension in the cord, and this incontinence may last for years, and yet the patient finally improve and obtain entire volitional control over the bladder again.

In other cases the resulting cystitis is an important factor in causing death.

Taylor and Lovett's 59 cases furnish 8 examples of paralysis of bladder or rectum, and the 138 cases show 63 examples.

(*g.*) *Reflexes.* The condition of the deep reflexes is important in diagnosis.[28 29] They are regularly exaggerated unless the seat of the lesion be the lumbar enlargement, in which case they are absent. The slightest tap on the patellar tendon, the lightest pressure on the sole of the foot, even a tap on the bone or fascia, or belly of muscle, will in many cases call forth a vigorous contraction of all the muscles of the leg, and sometimes be followed by a similar contraction of the muscles of the other leg. Even the evacuation of the bladder or rectum is said sometimes to be sufficient to call forth a clonus of the limbs.

The cutaneous reflexes are sometimes exaggerated, so that pinching the skin will cause a violent reflex clonus; again, are not to be found; and still again, are unaltered.

Gibney[15] quotes cases in which pressure over the inner side of the lower third of the thigh would cause sudden flexion of the thigh on the pelvis and of the leg on the thigh, followed in a few minutes by the same motion on the opposite side.

Bastian[28] says that cases show that "deep reflexes are abolished if there is a complete transverse lesion, *i. e.*, when there is a complete loss of sensibility as well as of voluntary power."

(*h.*) *Trophic and vasomotor changes.* Trophic changes are said to be uncommon unless the cervical or lumbar enlargement is involved, which may occur from secondary degeneration; but if the paralysis has existed any considerable time one frequently finds a notable degree of atrophy of the legs, together with some coldness, occasional lividity, and generally a dry, inelastic, scaly skin. There may be localized œdemas, sweatings, alterations of temperature,[18] and sometimes lesions of the joints secondary to lesions of the cord.

Roser[30] has seen three cases of spontaneous dislocation of the hip brought about by the reflex motions of Pott's paralysis; the luxation was behind, gradual and painless. In severe cases bedsores are frequent. Of the 138 tabulated cases, 20 had marked wasting of the limbs and 2 had profuse sweatings noted.[31]

(*i.*) *Medullary symptoms.* When the upper cervical cord is compressed Charcot has noted paralytic myosis or spastic mydriasis from paralysis or irritation of the corresponding paths in the cervical medulla.

Respiratory and gastric disturbances, vomiting, difficult swallowing, persistent hiccough, and retardation of the pulse have been met and are attributed to implication of the vagus, spinal accessory, or phrenic nerve; according to Michaud,[1] general convulsions may occur from compression of the upper cord.

(*j.*) *Sexual disability.* Pott speaks of a characteristic loss of the power of erection. In the series under examination priapism and loss of sexual power are mentioned, the former occurring three times and the latter twice.

(*k.*) *Time in Pott's disease at which paralysis appears.* The paralysis may come on as a diagnostic symptom of Pott's disease before the latter has been otherwise made manifest, or it may come on when spinal disease has existed for years and when to all appearances the process has subsided into quiescence.

Taylor and Lovett say paralysis occurs on an average about two years after the beginning of the disease. Out of 40 cases it came promptly after a fall in 4 cases; in 8 cases it came within one year from the beginning of the disease; in 16 cases within two years; in 4 cases within three years; in 3 cases within four years; in 1 case in five years; 1 in nine years; 1 in eleven years; 4 in fifteen years; and 1 in twenty-eight years.

Gibney says the average duration of spinal trouble before paralysis is about three years, though the time has been as short as four and a half months and as long as eleven years. Myers puts the time at from a year to a year and a half.

The writer has found the time in question in 130 cases as follows: Under one year, 62; one to two years, 18; two to three years, 14; three to four years, 10; four to ten years, 12; ten years or over, 14; longest time, twenty-one years.

From this it is apparent that in nearly 48 per cent. the time is less than a year, and that in more than 72 per cent. of the cases the time is less than three years.

(*l.*) *Duration of increase of paralysis.* From the first note of weakness to the maximum of paralysis attained there may be less than a day or there may be several months, but perhaps oftenest a few weeks.

In 44 cases the time was noted with the following result:

Under one month, 19 cases; one to two months, 12; two to three months, 4; over three months, 9.

(*m.*) *Duration of paralysis.* The duration of the attack may be a few weeks or it may be a lifetime; usually it varies from a few months to a few years. Pott[17] put the time at from two months to two years. Gibney[15] in 13 cases of paresis found a duration ranging from four weeks to twenty-two months. From a study of 218 patients, Myers[23] gives an average duration in cervical cases of twelve months, in upper dorsal of nine and a half months, in lower dorsal of six months, and in lumbar cases of eight months. He also finds that the duration is several months less when the paralysis begins in a patient under treatment.

Taylor and Lovett[22] give as the average duration twelve months, but when paralysis comes on while the patient is under treatment an average

duration of only seven months. In their 36 cases the durations were as follows: One case, six years; 2, three years; 5, two years; 7, one year; 4, six to twelve months; 8, six months: 9, two to four months.

The duration to recovery or death of 108 cases was as follows: Under six months, 37; six months to one year, 28; one to two years, 25; two to three years, 11; three to four years, 4; four or more years, 3. Twelve of these cases were under treatment before paralysis began. Of these 12 the duration was under six months in 6; six to twelve months in 5; and from 1 to two years in 1 case. The shortest paralysis with recovery noted by the writer is seven weeks and the longest four years.

(*n.*) *Recurrences.* A few cases have more than one attack, and there are instances of three, and even of four attacks.

Taylor and Lovett found in 59 cases recurrences in 6; Myers in 218 cases, recurrences in 18; Gibney in 58 cases, recurrences in 8. The intervals between these recurrences varied from a few weeks to several years.

Of the 125 patients studied 9 had two attacks, 1 three attacks, and 1 four attacks. The intervals between these attacks varied from a few weeks to fifteen years.

(*o.*) *Termination.* There are three terminations—recovery, death, and permanent paralysis.

1. Recovery is usually gradual, and frequently lasts months. After some time of absolutely no change in the patient's symptoms it is found that a few motions can be executed by the paralyzed parts at times, then there is a permanent increase of power in those parts; the sensibility improves and generally becomes normal before entire power over the muscles is established; control of the bladder is regained; and, last of all, usually months after the patient believes himself well, the tendon reflexes become normal.

2. Death may result from progressive myelitis with its attendant cystitis, bedsores, diarrhœa, pyæmic fever, and exhaustion, or the patient may yield to tuberculosis or be cut off by some intercurrent disease.

Gibney in 13 deaths found 6 from myelitis, 2 from tuberculosis, and 5 from other diseases.

Taylor and Lovett in 59 cases found 5 deaths—2 pneumonias, 1 phthisis, 1 opium habit, and 1 acute cerebral meningitis.

The list here studied furnishes 27 deaths with known causes; 8 were the direct result of myelitis, 8 were the result of tuberculosis or amyloid disease, 4 the result of operation for the relief of the paralysis, and the remaining 7 came from acute affections not bearing any relation to the spinal disease.

3. A permanent paralysis may be slight in amount—simply noticed as a weakness—or it may be a paralysis with contractures, and the patient get about, if at all, with great difficulty. In the last case, how-

ever, the general health may be excellent and the condition without danger to life.

FREQUENCY.—The proportion of cases of spinal caries that become paralyzed varies greatly with the clinic of the one making the estimate.

The 2657 cases of Pott's disease from the following table give 404 cases of paralysis, or about 15 per cent.

Authority.	Cases of spinal caries.	Cases of paralysis.	Per cent.
Gibney[15]	295	62	21
Taylor and Lovett[22]	445	59	13
Mohr[32]	72	5	7
Drachman[33]	166	22	13.6
Sayre[34]	109	38	35
Shaffer[23]	1570	218	13.9

In Gibney's cases, in 189 cases of cervical or upper dorsal disease, paralysis occurred 59 times, and in 106 of lower dorsal and lumbar there was paralysis only 3 times. Taylor and Lovett's 59 cases are distributed thus: 1 cervical, 7 cervico-dorsal, 37 dorsal, 7 dorso-lumbar, 4 lumbar, and 3 undesignated. Of Sayre's 38 cases, 2 were cervical, 1 dorso-cervical, 32 dorsal, 1 dorso-lumbar, and 2 lumbar. Shaffer's 218 cases are 16 cervical, 12 cervico-dorsal, 145 dorsal, 19 dorso-lumbar, 18 lumbar, and 1 unspecified.

In the present list the cases, so far as known, are apportioned as follows: Cervical, 15; cervico-dorsal, 10; dorsal, 85; dorso-lumbar, 6; and lumbar, 3. It is evident that nearly all the cases of paralysis result from lesions in the cervical or dorsal vertebræ, and that that locality of caries is, as Gibney says, the great predisposing cause of paralysis.

DIAGNOSIS.—Recognition of the beginning of paralysis is usually possible from the fact that it is generally preceded by an increase of pain in the abdomen or legs and by an increase in the deep reflexes of the knee and ankle. The diagnosis is made by establishing the existence of Pott's disease.

Trauma of the spine and pressure on the cord from other causes may give the same symptoms. Such other causes are cancer and sarcoma of the spine, small exostoses of the vertebræ, meningeal tumors, hypertrophic cervical pachymeningitis, aneurism, gumma, hydatid cyst, hemorrhage or abscess in canal, tumors of the cord, and spontaneous transverse myelitis.

When Pott's disease exists, the attendant asthenia or a double psoas contraction may be confounded with paralysis, but the latter has increased reflexes, and the former not.

PROGNOSIS.—In a broad way the prognosis of Pott's paralysis is good, *i. e.*, it tends to recover entirely. Taylor and Lovett in 47 cases, the results of which are known, find 39 recoveries (83 per cent.). Gibney

finds in 58 cases 29 recoveries (50 per cent.), and Myers reports 55 per cent. recovered in 218 cases.

Counting those 12 cases as "result not known," in which the paralysis has been in existence less than a year and still continues, the list here made gives 126 "result known" cases, of which 78 are recoveries (62 per cent.).

The Children's Hospital cases, considered separately, give out of 28 cases 18 recoveries (64 per cent.).

In making the prognosis in any particular case certain factors enter which may be considered in detail.

1. *Treatment of patient before paralysis.* If the patient has been under treatment, the prognosis is somewhat better. Taylor and Lovett give 100 per cent. of recoveries in such cases, but in the present list are 15 such cases, with only 10 recoveries (67 per cent.).

2. *Duration of paralysis.* The maximum time with recovery is, as before stated, four years, and of the 14 cases whose paralysis lasted more than three years only 4 recovered. In the cases whose paralysis lasted less than six months, there were 18 recoveries; 21 recovered in from six months to one year; 20 in from one to two years; and 11 in from two to three years. From this one may infer that a patient's recovery is more apt to come in the second six months than in the first, that it is nearly as likely to take place during the second year as during the last half of the first, but that the prognosis grows rapidly worse during the third year, and that after three years there is comparatively little hope.

3. *Time from beginning of spinal disease to beginning of paralysis.* Of 62 cases whose paralysis began within a year, 27, or 43.5 per cent., recovered, while of 68 whose paralysis began more than a year after the spinal disease, 44, or 64.7 per cent., recovered. In other words, if the process is acute and advancing rapidly enough to bring on a paralysis early, the prognosis is worse than if slower changes compress the cord at a later stage.

4. *Age of patient.* Of 15 cases over forty years old when paralysis began, only 5, or 33 per cent., recovered (the oldest cured was at beginning of the paralysis fifty-four years), while of 76 patients under fifteen years, 48, or 63 per cent., recovered. (The youngest cured was at the beginning of the paralysis twenty-two months old.)

Of 47 between fifteen and forty years, 24, or 51 per cent., recovered. Other things being equal, therefore, the prognosis betters with the youth of the patient.

5. *Severity of the paralysis.* Of the cases with simple paresis of the affected muscles, 65.6 per cent. recovered, and 15 per cent. died, while in those with complete paralysis of the affected parts, 36 per cent. recovered and 46 per cent. died; 38 per cent. of those with marked

sensory affection got well and 38 per cent. died; 41 per cent. of those with paralysis of sphincters recovered and 32 per cent. died.

Clearly, then, the lighter the paralysis the better the prognosis, but with complete paralysis the addition of sensory or vesical palsy does not make the chance of recovery materially less.

6. *Location of lesion.* Cases other than cervical and dorsal are too few to form a basis for estimate. The percentage of recovery in paralysis of cervical origin is 25, while in that of dorsal origin it is 55.

7. *Rigidity or contractures of limbs.* Of the 19 cases in which rigidity was mentioned as a prominent symptom only 5 recovered.

8. *Marked wasting of limbs.* Of 20 cases affected by considerable atrophy, 8 recovered.

9. *Character of spinal deformity.* The kyphosis, as was noted by Pott, is apparently not of great importance in prognosis, but a large gradual curve, as the writer found in thirty cases examined, claims the largest per cent. of recoveries.

10. *Sex.* As to the liability of paralysis in Pott's disease, it is interesting to note that of the 125 patients, 77 are males and only 48 females. The per cent. of recoveries among the men is 52 and among the women 61.

11. *Part of body involved.* When there is more than a paraplegia present, the prognosis is made correspondingly worse—7 recoveries out of 26 cases; while in simple paraplegia there were 71 recoveries in 112 cases.

12. *Recurrences.* The cases studied give no reason for thinking the prognosis in a second attack any worse than for a first attack.

13. *Intercurrent disease.* Bradford has noted a case of recovery from paralysis during an attack of measles. The writer has seen two cases of intercurrent typhoid; in one, as the fever progressed, the paralysis grew worse till the patient was powerless, and then lightened with convalescence; the other, a fairly severe case of paralysis, was quite unaffected by the fever. He has seen also one case of amyloid disease which died paretic; in this there was marked improvement in the paralysis during the two months immediately preceding death.

Proportion of deaths. Taylor and Lovett find in 59 cases 5 deaths, 3 still paretic. Gibney finds in 58 cases 13 deaths, 14 still paretic. In the 138 cases there were 34 deaths, 26 still paretic.

General summary of prognosis of paralysis. There is not a symptom characteristic of the paralysis that is of fatal import. Recovery has taken place even from threatening asphyxia.[35]

There is hardly a condition of the paralysis so severe as to deny the possibility of recovery. Recovery has occurred from paralysis of arms and legs, anæsthesia, and incontinence.

A child less than ten years old, with dorsal caries and paresis of legs for less than one year, will probably recover.

An adult paralyzed more than four years will probably not recover.

The three most important elements in prognosis, in order of their importance, are (*a*) the duration of the paralysis; (*b*) the severity and extent of the paralysis; (*c*) the age of the patient. The real index to the severity and extent of the paralysis is the completeness of the paralysis of the muscles of the affected limbs.

TREATMENT.—All authorities agree that the back should be put at rest; therefore confinement in bed, with a spinal support, either jacket or brace, must be the first measure adopted.

In certain cases this enforced rest, perhaps aided by extension and counter-extension, is sufficient for prompt cure, but in more cases cure is a matter of gradual improvement extending over months. In all cases rest should be persisted in till the return of the use of the limbs is well established. Relief in a few days or weeks has been given by opening an abscess connected with the spine, and operation is to be recommended if there is an abscess present. Subsequent treatment is expectant: the number of drugs given outside of tonics is large enough to prove that no one is eminently satisfactory.

Gibney[36] is confident that potassic iodide in doses to the limit of toleration reduces the average duration of the paralysis by months. He says: "Begin with gr. x, t. i. d. p. c. in mineral water, best Vichy; increase dose daily by gr. x till stomach is intolerant." "Keep up as big a dose as the stomach will bear."

Bromide, ergot, silver nitrate, strychnine, physostigma, belladonna, have all had earnest advocates, the last especially, for removing neuralgias which precede paresis. Outside of drugs, electricity is recommended especially by the neurologists, and Erb[2] says that a moderately strong galvanic current applied in a stabile way to the point of lesion has been of service, and that he has never seen it do harm. Gibney,[36] however, quotes cases where electrical treatment has, he says, caused relapse or delay in cure. The cautery is a favorite with some, but has objections for private practice. Althaus[37] says that this treatment is good when only the membranes are affected, useless if the cord is involved.

Poore[38] gives directions how to cauterize without causing notable suffering even in children, and cases are quoted in which a single cauterization has brought about an almost immediate cure.

In the same line of treatment are mentioned blisters and local applications of hot water and ice.

Mitchell[39] urges the claims of suspension, saying: "It lets the patient get out of bed; it distinctly alters the pathological curve of the spine; it gives ease and relief to aches; it seems to act with more speed than other plans, and when these have failed it gives a new resource."

Medicated baths are mentioned by one writer with the remark that they may be dangerous. Tenotomy[1] has been done to relieve the con-

tractures of paralysis. With reference to all the methods of treatment beyond rest, it is to be noted that as the paralysis tends to a cure which sometimes comes suddenly and unexpectedly, a careful estimate of the relative value of different remedies is difficult, and certainly from this series no conclusion can be drawn decidedly favorable to any mode of treatment beyond rest. Some cases, however, do not get well, and even grow worse very rapidly; for such there has been since 1882 another treatment proposed, resection of the vertebral laminæ at the site of the lesion and relief of presure. The operation has as yet been done only a few times, but there have been some remarkable successes and several fatal results; improvement when noted has frequently proved temporary. At present Bradford[40] says: "The status of operation is that it offers hope to some cases that would otherwise be hopeless, while it has no place in treatment till conservative measures have been faithfully tried."

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A CLINICAL AND EXPERIMENTAL STUDY OF URÆMIA.[1]

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Uræmia is a systemic intoxication, the phenomena of which are mainly nervous in origin, brought about by an accumulation in the blood of substances which it is the function of the kidneys to excrete, either by an over-production of such substances or by the failure of the kidneys to eliminate them owing to a lack of secreting power.

Etiology.—While it is probable that an appreciable number of cases of uræmia originate with kidneys in an apparently normal state structurally, yet Bright's disease is by far the most important factor in its development.

Though the total number of cases developing in the course of chronic Bright's disease is greater than that in the acute form, yet the percentage of uræmias in the latter is decidedly higher than in the former. In acute Bright's, most notably in scarlatinal nephritis, the onset of uræmia may occur at any time, even almost coinciding with the earliest positive symptoms of the kidney lesion.

In chronic nephritis uræmic symptoms will most frequently develop late in the disease, when the secreting substance of the kidney has been almost totally destroyed, or if occurring earlier, the attack will very frequently be found to coincide with an acute exacerbation of the chronic kidney lesion. This may be traceable, it may be, to exposure, exhaustion, or errors of diet. The influence of diet upon these exacerbations, and so upon the production of uræmia, is a subject that requires much more careful and systematic investigation than it has hitherto received, for while such influence is scarcely to be questioned, still no definite rules can be laid down governing it. It is a very significant fact that the simplest diet, of which milk is the best example, will obviate almost

[1] This essay was awarded the Boylston Prize, 1893, for the best dissertation on a Clinical and Experimental Investigation of Uræmia.

By an order adopted in 1826, the Secretary was directed to publish annually the following votes: 1. That the Board do not consider themselves as approving the doctrines contained in any of the dissertations to which premiums may be adjudged. 2. That in case of publication of a successful dissertation, the author be considered as bound to print the above vote in connection therewith.

The Boylston Medical Committee is appointed by the President and Fellows, and consists of the following physicians: Robert T. Edes, M.D., President; Samuel G. Webber, M.D.; H. P. Bowditch, M.D.; Frank W. Draper, M.D.; J. Collins Warren, M.D.; Edward S. Wood, M.D.; William F. Whitney, M.D., Secretary; F. H. Williams, M.D.

wholly these accidents. Of errors in diet, alcoholic excesses stand easily at the head. Such a notable number of cases of uræmia occur in the course of acute alcoholism that there can be no question of the relation of cause to effect. Many symptoms of acute alcoholism are probably uræmic, even though there may be no pronounced kidney lesion demonstrable. The alcohol probably plays a dual rôle: first, by its irritant action upon the gastro-intestinal mucous membrane it causes an increased production of toxic substances; and second, after its absorption, its direct effect upon the kidney must be such as to perceptibly limit elimination.

Many cases occurring in the course of chronic nephritis are indeterminate in etiology. Particularly is this true of the interstitial form when uræmia comes on suddenly, without any indisposition or apparent cause. In the parenchymatous form serious uræmia will most likely occur very late, and with fatal result; or if earlier, it is almost always traceable to an exacerbation of the nephritis, and may be recovered from. Occurring in the course of Bright's disease, uræmia is usually preceded or accompanied by a decreased secretion or total suppression of urine. Too much attention, however, cannot be drawn to the fact that well-marked and characteristic uræmia may occur while the quantity of urine is not at all disturbed. Further, it is to be noted that there may be uræmia while the urine is not at all albuminous, even when post-mortem examination has disclosed well-marked disease of the kidneys.

Again, if during the course of a chronic nephritis there be an intercurrent acute febrile affection, this often determines the onset of uræmia.

In addition to actual diseased conditions of the kidneys, there is a class of cases where, though there be a healthy state anatomically present, yet functionally they are inactive through changed urine pressure, and the same result is attained as by disease. These cases are the uræmias occurring during pregnancy or other conditions which interfere with the free outflow of urine. Here often two factors may be recognized, one of them a somewhat diseased condition of the kidney, yet it is undeniable that in some of the cases only the other factor obtains—obstruction of the ureters and damming back of the urine on the kidneys. Several such cases have been recently reported by Nash.[1] While here many of the eclampsias are unquestionably the concomitants of definite kidney lesions, as is evidenced by the accompanying urinary condition, yet there are some occurring with perfectly normal urine, in which the kidney can only be functionally inactive. This is rendered the more probable, seeing that in the lower animals, where there can be no pressure by the pregnant uterus upon the ureters, eclampsia is unknown. There is a class of cases where the symptoms are not so clearly uræmic, and even if uræmic, where the etiology is certainly more obscure than in

[1] Lancet, London, 1892, vol. ii. pp. 477.

the foregoing. These are the so-called uræmias of the acute infectious diseases. The condition is most marked, and has received the greatest attention in cholera, but is also encountered in typhus and typhoid fevers, and probably rarely in some other diseases. Here the symptoms are so exactly similar to the uræmia occurring in Bright's disease that they are commonly referred to as uræmic. Of course, in some of these cases there is actual disease of the kidneys, and here the condition is really one of uræmia due to loss of secreting power of the kidneys, the direct consequence of their diseased condition. But, when the kidneys are structurally almost perfect, the uræmia, if it really be uræmia, must be due to excess of poisonous matters in the blood, either on account of an actual over-production or else to lack of elimination by the kidneys, either purely functional or due to the changed condition of the blood, the result of the cholera. These cases, it seems to us, instead of being considered uræmias, would better be classed among the toxæmias, the toxines producing them being the products of the specific bacteria causing the diseases. However, they may really be uræmic, and due to an over-production in the blood of the uræmic poison, seeing from the consideration about to be entered upon that such a condition seems to be possible.

In health, with the kidneys structurally perfect, as is evidenced by the urine, it is possible to have uræmia develop, which is probably, at least in part, to be explained by the assumption of an over-production of poison.

CASE: Man, aged forty-three years; always in fairly good health, with the exception of rather marked dyspepsia. Without cause or warning he was suddenly seized with a general convulsion, attended with entire loss of consciousness. When first seen, some hours later, he had recovered from the convulsion, but was delirious. It was impossible to attract his attention, or elicit any coherent answers to questions. For twenty-four hours there was no urine voided, and a catheter passed into the bladder found it empty. At the end of this time the secretion of urine was slowly re-established. The first urine that passed, three ounces, had a specific gravity of 1033, contained no albumin and no casts, but an excessive amount of uric acid. As the urine increased in quantity the specific gravity was slightly lowered, but it never fell below 1028. There was constantly present an excess of uric acid, but at no time could either albumin or casts be detected by a most careful examination. The delirium persisted for a week, gradually subsiding. Since then, there has been no return of any such symptoms. Repeated examinations of the urine for four years after the convulsion, failed to show either albumin or casts, nor has there been any other evidence of renal lesion. The urine has always been above the average normal specific gravity, and has generally contained an excess of uric acid. No examination of the urine was made previous to the convulsion.

Now, while here were presented none of the ordinarily accepted causes of uræmia, the case is explicable on no other grounds than that

the symptoms were uræmic, or at least due to a poison acting similarly to the uræmic poison and producing similar results. It is probable, looking at the pronounced dyspeptic symptoms and the marked excess of uric acid, that there was an excessive production of poison in the digestive or assimilative apparatus. It is impossible that the symptoms could have been due to any disease of the central nervous system, for a most careful analysis of the case has failed absolutely to point to any such disease. Two other cases of probably similar character have been under observation, but the symptoms have been much less marked.

Such conditions as these must not be confounded with uræmia arising in the course of Bright's disease, when the urine is non-albuminous, the possibility of which has been mentioned above. It is perfectly well known that the interstitial form of chronic Bright's disease may exist without the presence of albumin in the urine, and that uræmia may arise in such a condition; but it is apparently not recognized that such a state of affairs may obtain equally in the parenchymatous form. Dr. Leys[1] has drawn attention to this fact with the citation of the following case:

A man, aged fifty-one years, of alcoholic habits, had been at the Philadelphia Hospital in 1885 with nephritis, from which he apparently recovered and was discharged. On March 21, 1892, he became suddenly insane, and was readmitted. On admission he was deeply stuporous, could be aroused to say "yes" and "no;" legs œdematous; breath distinctly urinous in odor; pupils strongly contracted and immobile; temperature 99.4° F.; urine had a specific gravity of 1016 and contained neither albumin nor casts. In the next twenty-four hours the patient roused somewhat, temperature fell to normal, pupils became normal, and thirty-six ounces of urine was passed, containing no albumin. In the following twenty-four hours he grew perfectly rational, but had no recollection of preceding events; the œdema disappeared and the urine rose to forty-five ounces. November 25th, the urine fell to thirteen ounces, and all the bad symptoms except the œdema reappeared. Then the patient made a slight rally; but the urine, which still showed no trace of albumin, did not increase. November 27th, the temperature rose to 100° F., and the patient died of pulmonary œdema. Urine drawn from the bladder after death showed albumin distinctly, and two granular casts were found after a prolonged search. Post-mortem examination showed the kidneys large (right weighed eight and one-half ounces, left ten ounces), capsules distinctly adherent, the surface and section a light, mottled pink, cortex much thickened and pale, pyramids congested. The heart showed fatty degeneration, but was not hypertrophied. The brain showed no lesions except œdema of membranes.

SYMPTOMATOLOGY.—For clinical study uræmia may be divided into the acute and chronic forms. The conditions under which these forms appear are the same, and no sharp line can be drawn between them.

[1] Philadelphia Hospital Reports, vol. ii.

The symptoms are mainly the same, differing only in severity. At any time during the course of chronic uræmia exacerbations may supervene as violent as any of the acute cases. So that, in considering any symptom it must be premised that it is limited to neither form. The acute form is usually sudden in its onset and well marked in its course. It is most likely to be met with in acute, or in the exacerbation of chronic, Bright's disease. The chronic form arises more gradually, and, lasting indefinitely, is present toward the end of a chronic kidney affection. It may be recovered from, and an indefinite interval of comparative health be enjoyed, or more usually it leads surely to death, its close being marked by a violent outbreak. The symptoms may be distributed among the nervous, the gastro-intestinal, and the cardio-pulmonary systems; but symptoms of one type may be intermingled with those of another, or at any time one type may be replaced by another.

Nervous System.—One of the commonest, and certainly the most marked, manifestations is convulsions, sometimes preceded for a short time by headache, vertigo, nervousness, muscular twitchings, nausea, or vomiting, and sometimes with an abrupt onset. They resemble exactly epileptic convulsions, lacking the aura and the initial cry. Lasting a variable time, they are followed by somnolence or mental heaviness, and may recur indefinitely. Sometimes the convulsions follow each other in rapid succession till they end in death. Or, a convulsion may terminate in coma, persisting till death or eventually clearing up. More rarely, instead of coma, delirium or actual mania may follow a convulsion. Usually general, the convulsions may be unilateral, or even limited to a single member; here consciousness need not be lost, or only for a period so brief as not to be clearly recognized.

The pupils in the convulsions are a subject of some dispute. It may be said that no rule can be laid down here; usually normal or slightly contracted, they may be widely dilated and occasionally they are unequal. Whatever may be their condition of dilatation, they are most frequently slightly susceptible to the influence of light, even in the height of the convulsion. In that condition in which convulsion follows convulsion in rapid succession they are most likely to be dilated.

Muscular twitchings are sometimes the harbinger of a convulsion, or they may come and go for an indefinite time, even without the existence of any other uræmic symptoms, though they are most frequently associated with a certain amount of headache or mental hebetude. Coma, as has been said, may follow a convulsion, or the convulsion may be succeeded by delirium, which in turn gives place to coma. Usually there is first a certain amount of mental hebetude, which is accompanied by headache, muscular twitchings, or other slight uræmic symptoms. This deepens—after lasting for hours, days, or even weeks—through

somnolence into coma. Sometimes the coma, after it has become well established, is interrupted by violent convulsions. Somewhat rarely coma appears suddenly as the first symptom, and in this case it may resemble strikingly cerebral apoplexy. As apoplexy has one of its common causes in the arterial degeneration of Bright's disease, there is much danger of confounding such a condition of sudden uræmic coma with it; but a careful examination, when the condition is uræmic, will show merely a general flaccidity of the muscular system and not any localized paralysis; or if there be paralysis, it will be much slighter than could be expected with the marked cerebral symptoms. The course of the case will clear up the diagnosis, if there be doubt in the beginning; in uræmia, if there be any slight paralysis, it will not deepen; while the coma, if it persists, will generally develop into a marked typhoid type. The condition of the pupils is not of any positive value, for in uræmia they may be distinctly unequal, though this is exceptional.

The diagnosis between the coma of alcoholism and uræmia is even more difficult. Alcoholism in the course of Bright's disease is one of the most frequent exciting causes of uræmia. The fact that there is an acute alcoholism present should always arouse a suspicion of the possible presence of uræmia. Aside from the history of the onset, which is the most important when it can be obtained, the best diagnostic points are that alcoholic coma is scarcely so profound nor is it so frequently associated with convulsions. Prognostically, coma is the most important of uræmic symptoms, for it is the one which most frequently announces oncoming death.

Headache is one of the very common symptoms of uræmia. Very early it is likely to be transient, occurring probably only in the morning. As the case progresses and other symptoms are added it becomes more intense and persistent, sometimes being so violent as to be agonizing. Not limited to any particular part of the head, our experience has been that it is most frequently frontal, while Dr. Osler[1] states that occipital headache is most common. Occasionally it resembles migraine, limited to one side of the head, usually frontal, excruciatingly severe, paroxysmal, and often attended by vomiting. Headache is not rarely the symptom which ushers in a grave attack of uræmia. Vertigo is frequently associated with the headache.

Neuralgias frequently occur early in the course of uræmia and often in the less marked cases. They may occur in the course of any nerve, but are most common in the distribution of the fifth cranial nerve, assuming generally the type of hemicrania or facial neuralgia. They are not nearly so likely as headache to be associated with more pronounced

[1] Practice of Medicine, p. 739.

uræmia, and it is probable that the anæmia which is present plays as important a part as the uræmic poison in their etiology.

Occasionally formication, the sensation of pins and needles, or localized vasomotor effects, as, for instance, a condition of the fingers such as is found in Raynaud's disease, have been noted, and are probably uræmic, from the fact that they are associated with more definite symptoms. The formication is most frequently unilateral or limited to a single member. It is worthy of note that similar symptoms are found in dyspepsias, especially those of gouty character. A troublesome itching, sometimes general, sometimes confined to the genitals, is occasionally complained of. It is likely to be a late symptom and associated with the typhoid state, coming on at the close of a prolonged uræmia.

Localized palsies are of infrequent occurrence. They may undoubtedly be purely uræmic in origin, especially so when they are transient and follow a convulsion, or occur in the course of only slightly marked cerebral symptoms. When associated with coma or persisting it is questionable whether they are purely functional and not due to some slight pathological changes in the brain substance. Post-mortem examination of brains, when such paralyses have persisted for some time before death, always shows some change, often a localized one, either œdema, congestion of the meninges, or punctiform cortical hemorrhages. The fact that such paralyses occur usually in cases where there are widespread arterial changes, and when there have often been symptoms of brain involvement other than those unquestionably uræmic, would lend weight to the belief that though uræmia may be the actual exciting cause, yet the anatomical condition of the brain determines the exact symptoms. Acheson[1] has found, in addition to the gross lesions, microscopical changes in the cerebral matter. Delirium is a symptom of not very frequent occurrence, but so characteristic when it does occur that its significance cannot be too highly appreciated. Unless its importance be kept in view it may be misinterpreted, and a failure to make a correct diagnosis ensue. Usually it is of a mild talkative character, and it may persist for an indefinite time. A case typically illustrative has been under observation since January 27, 1893, and is briefly as follows:

CASE: Negro, man, aged fifty-one years; when first seen was passing twenty ounces of urine in twenty-four hours, heavily loaded with albumin and containing casts in abundance. Talkative—even noisy at times—wandering from subject to subject rapidly, and in no connected sequence, but always having most prominent religious subjects; it was impossible to obtain from him any reliable data as to the origin of his attack. Eventually he developed distinct delusions of a religious nature. During this period he had muscular twitchings, most marked in the upper

[1] Canadian Practitioner, vol. xiv, p. 125.

extremities. This state lasted two days, when it was interrupted by a marked convulsion, whereupon he was bled. A very trifling convulsion followed a few hours later on. After that the original delirium was re-established, and has since persisted.[1]

Sometimes the delirium, especially in bad types of cases, is low and typhoid in form, and is attended with the general symptoms of the typhoid state. Here an early and unfortunate termination is to be feared. At any time, any form of delirium may be interrupted by maniacal outbursts, which are frequently precursors of convulsions, or the delirium may drift gradually into coma.

Closely associated with delirium is uræmic insanity, which may arise in the course of delirium, or without premonitory symptoms may supervene with startling suddenness. Though by no means common, its possible existence must be recognized. The forms ordinarily encountered are, in the order of their frequency, acute mania, delusional insanity, and melancholia. It is possible to have almost any type of insanity as the result of uræmia. Puerperal mania will not rarely be found to be uræmic.

CASE: A young woman pregnant with first child while in labor was, without premonitory symptoms, seized with a convulsion. This was relieved by free bleeding, but she went from the convulsion into a condition of most marked mania. This lasted for a week, when she began to grow rational, recognizing those about her. It was a month after the convulsion before her mind became fully restored. She had afterward no recollection whatever of her labor and subsequent mania, and only remembered very vaguely events which happened during her convalescence. The urine had not been examined before labor, but the first passed after the convulsion was loaded with albumin and contained numerous casts. By the time sanity was perfectly re-established albumin and casts had disappeared. Two subsequent pregnancies were attended with no demonstrable kidney lesion.

The delusional insanity is illustrated by the following:

CASE: A man aged forty-seven years, of alcoholic habits, had been dropsical since April, 1890. First came under observation in July, 1890, with general anasarca, diminished quantity of urine containing much albumin, some epithelial and a few oil casts. August 15, 1890, quantity of urine greatly diminished, and patient became delirious and noisy. Had the fixed delusion that his brother-in-law was about to kill him. The delirium cleared up entirely at times, but the delusion lasted throughout the disease—a period of three months. Died November 16, 1890. Autopsy showed typical chronic parenchymatous nephritis, with beginning contraction.

Of the special senses, sight and hearing are sometimes affected. Amaurosis, not a common symptom, is usually sudden in its onset and

[1] This condition lasted till death, late in April.

may be the first evidence of uræmia. It is not very grave prognostically, usually disappearing after a short time, ofttimes as suddenly as it came. Purely functional, no peculiarity of the eye-ground to account for it can be seen on ophthalmoscopic examination. An œdematous condition of the retina has been described, but it is more likely due to centric disturbance. In considering amaurosis as a symptom of uræmia it must be borne in mind that vision may be affected by albuminuric retinitis, and the ophthalmoscope must be used to clear up all doubt.

Deafness, resembling in its onset and course amaurosis, is said to be a uræmic symptom; but of this we cannot speak authoritatively, having never seen a case.

Gastro-intestinal.—A buccal and pharyngeal form of uræmia has been described and attributed to an effort at elimination of the poison by the salivary glands and mucous membrane of the mouth and pharynx. It seems to us that these so-called symptoms are merely part of the exhausted or even typhoid state into which victims of Bright's disease may fall. Salivation may be, however, an actual symptom of uræmia, as we shall show in the course of our experiments. It is probably centric in its origin. While we have noted a pretty free flow of saliva in a few cases, but never except in pronounced uræmias, we consider this symptom of little importance clinically. It may be worthy of note that in our experience mercurial salivation is more frequent in Bright's disease than when the kidneys are healthy.

Vomiting is rather a common symptom. It is likely to come on early and may often be the precursor of a convulsion or other grave uræmic symptom. The matters vomited are first ingesta, then mucus, often bile-stained; nausea is rare and ingestion of food has little influence. The vomiting comes on suddenly and may persist an indefinite time, often ceasing and recurring irregularly. In the more chronic cases vomiting is often persistent and intractable. Here the matters vomited may be strongly alkaline in reaction, due to the presence of carbonate of ammonia. The carbonate of ammonia is produced by decomposition of urea excreted by the mucous membrane of the stomach from the blood. In these cases the vomiting is not centric, but due to a direct irritation of the stomach by the carbonate of ammonia. In other cases the vomiting is probably centric, as our experiments will show. There is in some cases a swollen or dropsical condition of the mucous membrane, as pointed out by Bartels. In considering vomiting as a symptom of uræmia, it must be remembered that in Bright's disease there is very generally a marked disturbance of the stomach as a part of the universal depression of tone of the system.

Diarrhœa, not so common as vomiting, is usually due to irritation by carbonate of ammonia, and is less likely than vomiting to occur early. Sometimes the irritation is so excessive and prolonged as to lead to an

actual inflammation or ulceration of the intestinal mucous membrane, attended by the presence of blood in the stools. Vomiting and diarrhœa seem to be eliminative, for when they are present nervous symptoms are uncommon, and nervous symptoms may even cease upon the establishment of a sharp diarrhœa.

CARDIO-RESPIRATORY.—The heart's action is seldom definitely disturbed. Occasionally, preceding a convulsion, the pulse is slowed; more often it is accelerated, but so inconstant is any such disturbance that it is of no diagnostic value. Rarely uræmia seems to be productive of tachycardia.

CASE: A woman, forty-four years old, the subject of chronic interstitial nephritis. No demonstrable arterial changes. Pulse persistently rapid, never falling below 100 and often irregular. Had for some time trifling uræmic symptoms, and finally went into a condition of pronounced uræmia, with active delirium, alternating with stuporous intervals. At this time the pulse increased in frequency, ranging between 160 and 180. After the uræmia subsided the pulse slowed somewhat, varying, as before the uræmic attack, between 100 and 140.

Occasionally uræmic attacks will be accompanied by violent palpitation.

Dyspnœa is an important symptom and one that is present not rarely. It shows itself in two forms: constant shortness of breath and paroxysms of asthma. In the first form the shortness of breath shows itself in the beginning only on exertion, afterward becoming constant and so marked that the patient is unable to lie down. It differs very little in its symptoms from other dyspnœas, though, as a rule, when the condition becomes well marked the respirations are rather full than shallow. Many cases of dyspnœa in the course of nephritis are really not uræmic, but can easily be explained by the physical condition of the lungs or heart.

Asthma is rarer than simple dyspnœa. The attacks resemble exactly bronchial asthma, and are most frequently nocturnal. Physical examinations will show a much-prolonged expiration and possibly bronchial râles. Occasionally these asthmatic attacks are interchangeable with other uræmic symptoms.

CASE: Man, aged sixty-six years; had been under observation for about a year. Urine contained albumin and casts. Some arterial sclerosis. On two occasions, after a short period of mental hebetude, there was developed a slight loss of power on the right side. Sensation was not interfered with, nor was there any affection of speech. On both occasions power was fully restored after the lapse of a few days. Finally, after similar mental symptoms, a paroxysm of asthma came on. In the next three days he had a number of asthmatic attacks, and died in one of them. Physical examination during the paroxysms showed slight pulmonary congestion, with prolonged harsh expiration. During the interval no abnormality in the lungs could be detected. The physical signs were not bilateral, sometimes one lung, sometimes the other, being their seat.

Cheyne-Stokes respiration is only noted in exceptionally severe cases, and usually those of a comatose type. It is a very grave symptom, and the cases in which it occurs usually terminate fatally, though exceptionally recovery may occur.

CASE: Man, aged fifty-five years; epileptic. Urine contained albumin and hyaline and granular casts. In July, 1890, had first attack of uræmia, from which he recovered. In November, 1890, became comatose again, and remained so several days. During the attack, Cheyne-Stokes respiration was present for nearly two days. Recovered from this attack, and in March, 1891, developed uræmia again and died. Cheyne-Stokes respiration was also present in the last attack. Autopsy showed a very small left kidney (probably non-development). Right kidney showed chronic parenchymatous degeneration with considerable contraction, and a few scars, apparently the result of infarcts.

During any uræmia the breath has frequently a urinous odor, and so characteristic is this that it is of great importance diagnostically.

TEMPERATURE.—The temperature of uræmia is not a fixed symptom. It may be normal, or above or below. In a convulsion the temperature is likely to be high, just as with any other convulsion; but without any convulsion there are some cases in which the temperature is persistently high even when there is absolutely no inflammatory lesion to account for the pyrexia. Is this high temperature an integral part of uræmia? It occurs in the presence of other symptoms unquestionably uræmic, may abate coincidentally with the subsidence of uræmia, and is the result of no demonstrable lesion, so that we are forced to the conclusion that it is really part of uræmia. There is really nothing theoretically to forbid such an assumption. Uræmia being due to a poison acting upon the central nervous system, the poison may easily disturb the heat-centres. Of the exact mechanism of the production of the fever it is impossible in the present state of our knowledge to speak at all confidently. This persistent high temperature, while rare, is sufficiently common to be of importance, and the literature of the subject for the past few years contains numerous examples. The following table shows the condition of the temperature in forty cases of uræmia. The cases were selected from a large series observed as containing no element other than uræmia that might disturb the temperature, and in every one of them the diagnosis was verified by a post-mortem examination. When the only temperature given is that in the column of averages, the variations were so slight as to be insignificant, and merely the average is noted.

Interstitial Nephritis.

Number of case.	Sex.	Extremes of temp.	Average temp.
1	Male.	98.0° to 101.2° F.	98.8° F.
2	"	96.4 " 98.0	97.4
3	"	98.0 " 99.4	98.7
4	"	— —	98.8
5	"	— —	96.6
6	"	— —	98.3
7	"	96.4 to 98.0	97.5
8	"	98.4 " 99.6	99.0
9	"	— —	97.9
10	"	— —	99.0
11	"	— —	98.4
12	"	— —	97.1
13	"	97.0 to 99.5	98.4
14	"	95.0 " 98.0	96.8
15	"	96.0 " 100.0	98.0
16	"	97.0 " 100.0	98.3
17	"	97.0 " 99.8	98.3
18	"	97.6 " 98.6	98.0
19	"	98.0 " 99.8	98.9
20	Female.	98.0 " 100.2	99.1
21	"	96.4 " 98.0	97.1
22	"	94.0 " 101.6	99.5

Parenchymatous Nephritis.

Number of case.	Sex.	Extremes of temp.	Average temp.
23	Male.	98.0° to 101.0° F.	99.4° F.
24	"	100.6 " 104.3	101.8
25	"	— —	97.4
26	"	— —	97.2
27	"	— —	96.8
28	"	99.3 to 102.0	101.9
29	"	— —	99.0
30	"	96.8 to 98.8	97.6
31	"	98.2 " 99.0	98.6
32	"	— —	97.2
33	"	— —	97.1
34	Female.	— —	97.9
35	"	100.0 to 103.8	102.0
36	"	98 0 " 101.0	99.0
37	"	97.0 " 100.8	99.2
38	"	— —	98.8
39	"	— —	98.2
40 (Pyelo-nephritis)	"	97.6 to 100.0	98.4

From this it will be seen that no rule can be laid down for the temperature. In considering the effect of the variety of kidney lesion only one fact can be obtained which is important—high temperature is more common in parenchymatous nephritis. The temperature was rather more frequently below than above normal, but the difference is scarcely so striking as to be of much value. It is to be noted, too, that in some cases the range was quite extensive, varying between distinctly

subnormal and pyrexia. The variety of uræmia present does not seem to have any constant effect. When the range was great the highest temperature was rather to be found toward the end than in the early stages. The following experiments upon dogs were undertaken with the object of determining the effect upon temperature produced by the introduction into the blood of substances supposed to be active in the production of uræmia. The temperatures are given in Centigrade.

EXPERIMENT I.—Dog, weight 18 pounds.

Time.	Blood pressure.	Pulse. (10 sec.)	Respiration. (30 sec.)	Temperature.
11.55 A.M.	122 mm.	27	22	39.4° C.
12.05 P.M.	Injected per jugular vein 10 c.c. human urine.			
12.05½	68	28	19	—
12.08	10 c.c. human urine.			
12.09	72	30	16	—
12.15	122	23	17	—
12.16	10 c.c. urine per jugular.			
12 26	116	23	14	37.5
12.26½	10 c.c. urine.			
12 27	82	25	Irregular.	—
12.28	116	20	16	—
12.34	10 c c. urine.			
12.35	96	22	16	—
12.41	114	22	16	—
12.42	10 c.c. urine.			
12.49	114	22	14	37.4
12.50	10 c.c. urine.			
12.57	110	27	—	—
12.57½	10 c.c. urine.			
12.58	94	28	15	—
1.04	112	24	—	—
1.04½	10 c.c. urine.			
1.06	92	26	16	—
1.10	104	24	—	—
1.10½	10 c.c. urine.			
1.11	86	27	—	—
1.15	10 c.c. urine.			
1.16	90	27	18	—
1.23	100	25	—	37.0
1.23½	10 c.c. urine.			
1.25	90	27	21	—
	Animal killed.			

EXP. II.—Dog, weight 35 pounds. Practically a repetition of Experiment I., in which 285 c.c. of human urine was injected intravenously. The injections were made about every five minutes in quantities of 10 c.c., 15 c.c., and 20 c.c. No effect was produced other than a fall of temperature of 1.4° C. in 1½ hours. The pressure was lowered somewhat immediately after the injection, soon returning to the normal, and was not affected as much toward the end of the experiment as it was in the beginning.

Exp. III.—Dog, weight 12 pounds.

Time.	Pressure.	Pulse.	Respiration.	Temperature.
11.25 A.M.	154 mm.	32	42	39.3° C.
11.28	Injected per jugular vein 20 c.c. of 20 per ct. sol. of *urea*.			
11.29	158	24	24	—
11.40	170	15	—	—
11.40½	Injected 20 c.c. of 20 per cent. solution of urea.			
11.41	166	15	—	—
12.06 P.M.	184	21	20	38.0
12.07	Injected 20 c.c.			
12.08	186	20	—	—
12.26	150	19	15	—
12.27	Injected 20 c.c.			
12.28	164	18	15	37.3
	Urinated very freely.			
1.15	140	20	—	37.3
2.25	Ligated right renal artery.			
2.26	156	18	—	37.5
	Animal killed.			

Exp. IV.—Dog, weight 20 pounds. Practically a repitition of Experiment III., in which 140 c.c. of 20 per cent. solution of urea was injected into the vein in the course of two hours. No effect was produced other than a fall of temperature of 1°and a profuse diuresis. At the end of two hours both renal arteries were tied and the observation continued for 1½ hours, but no change in the condition of the animal resulted.

Exp. V.—Dog, weight 46 pounds.

	Time.	Pressure.	Pulse. (10 sec.)	Respiration. (30 sec.)	Temperature.
Nov. 21st.	1.40 P.M.	100 mm.	21	30	40.5
	2.15	Right renal artery tied, antiseptically.			
	3.00	110	22	42	40.2
	Walks all right when untied.				
Nov. 22d.	12.12 P.M.	106	23	—	40.5
	12.25	Left artery tied.			
	1.08	86	21	33	40.4
	Animal quite dull.				
	2.00 P.M.	60	22	26	40.4
	2 45	60	23	—	40.4
	Seems very sick.				
Nov. 23d.	11.00 A.M.	Dog very sick.	—	—	39.0
	5.30 P.M.	—	—	—	40.0
	Animal died during night.				

Exp. VI.—Dog, weight 14 pounds.

	Time.	Pressure.	Pulse.	Respiration.	Temperature.
Nov. 26th.	2.00 P. M.	116 mm.	32	—	39.7°
	2.10	Injected per jugular 10 c.c. dog's urine.			
	2.11	52	36	—	—
	2.16	92	26	—	—
	2.16½	10 c.c. dog's urine.			
	2.17	44	27	—	—
	2.25	84	25	—	—
	2.25½	10 c.c.			
	2.26	46	28	—	39.5
	2.33	80	23	—	—
	2.33½	10 c.c.			
	2.34	54	32	—	—
	2.40	74	27	—	—
	2.40½	10 c.c.			
	2.41	48	31	—	39.5

Injections were continued in this way (10 c.c. at a time) until 100 c.c. had been given in all at 3 P.M. Pressure fell after each injection as above. No other effect.

	Time.	Pressure.	Pulse.	Respiration.	Temperature.
Nov. 26th.	5.30 P.M.	90 mm.	—	—	39.8° C.
Nov. 27th.	11 A.M.	Dog very sick.	—	—	40.0
	2 P.M.	46	19	—	—
	2.19	Injected 10 c.c. dog's urine per jugular.			
	2.19½	18	11	—	—
	2.20	Dog died.			

Exp. VII.—Dog, weight 18 pounds. Injected intra-venously 120 c.c. dog's urine, in doses of 10 c.c., at intervals of five minutes. The pressure was lowered greatly immediately after each injection, and two hours after the last injection was only half the normal. The pulse and respiration were slowed considerably and the temperature fell 2° C. Nov. 29th, temperature was 1.5° above normal all day. Dog died during night.

Exp. VIII.—Dog, weight 15 pounds. Injected into jugular vein 20 c.c. dog's urine in doses of 5 c.c. every ten minutes. The temperature was reduced about 1° C., but had returned almost to the norm four hours after the last injection. Next day injected 8 c.c. in doses of 2 c.c. every half-hour with practically no effect on the temperature.

Exp. IX.—Dog, weight 19 pounds. Injected subcutaneously 8 c.c. dog's own urine without any effect on the temperature.

Exp. X.—Dog, weight 18 pounds. Injected 5 c.c. dog's urine subcutaneously. No effect.

Exp. XI. and XII.—Dog, weight 15 pounds. Injected dog's own urine subcutaneously without producing any effect.

Exp. XIII.—Dog, weight 17 pounds. Injected subcutaneously 5 c.c. urine from a case of Bright's disease passing f℥xx per diem. Observed four hours after injection. Temperature fell 0.6° C. in three hours.

Exp. XIV.—Dog, weight 14 pounds. Injected subcutaneously 2 c.c. same urine as in Experiment XIII. Had no effect.

Exp. XV.—Dog, weight 17 pounds. Injected subcutaneously 4 c.c. urine from same case Bright's disease as in Experiment XIII. Temperature fell about 1° C. in two hours.

Exp. XVI. and XVII.—Injected subcutaneously 0.16 grm. and 0.8 grm. kreatin. No effect at all upon the temperature.

Exp. XVIII —Injected slowly 0.12 grm. kreatin into jugular vein. No effect upon pressure, pulse, or respiration. Temperature fell about 1.5°, but animal was tied down all the time.

Exp. XIX.—Injected 0.2 grm. kreatin without any effect.

Exp. XX.—Dog, weight 15 pounds. Injected slowly into jugular vein 2.48 grm. of kreatin with absolutely no effect on pressure, pulse, or respiration. The temperature fell some, but dog was tied down for about an hour and a half.

Exp. XXI and XXII.—Injected 0.1 grm. kreatinin subcutaneously in each experiment without any effect.

Exp. XXIII.—Dog, weight 24½ pounds. Injected into jugular 0.22 grm. kreatinin without any effect on pressure, pulse, respiration, or temperature.

Exp. XXIV.—Dog, weight 15 pounds.
3.45 P.M. Normal temperature 38.9° C.
4.30 Injected per jugular vein 50 c.c. blood serum[1] from a case of uræmia with hyperpyrexia.

[1] Blood taken with all precautions as to antisepsis, so that bacterial contamination did not take place.

4.45 Temperature 39° C.
4.55 Injected 50 c.c. blood serum.
5.15 Injected 50 c.c. blood serum.
5.20 Temperature 39.7° C.
5.30 Injected 50 c.c.
5.45 Temperature 39.4° C.
6.00 Temperature 39.4° C.

Exp. XXV.—Dog, weight 15 pounds.
3.40 P.M. Normal temperature 38.9° C.
4.15 Injected per jugular vein 50 c.c. blood serum from same case as in Experiment XXIV., heated to 58° C.
4.40 Injected 50 c.c.
4.50 Temperature 38.8° C.
5.10 Injected 30 c.c.
5.18 Temperature 38.4° C.
5.40 Temperature 38.2° C.

Exp. XXVI.—Large white rabbit.
4.00 P.M. Normal temperature 39.6° C. Injected subcutaneously 25 c.c. of blood serum from a case of uræmia without pyrexia.
4.40 No effect apparent.
5.00 Injected 50 c.c. subcutaneously. Rabbit lying on side and breathing very rapidly.
5.30 Temperature 37.5° C.

Exp. XXVII.—Black rabbit.
4.00 P.M. Normal temperature 39.8° C. Injected subcutaneously 25 c.c. same blood serum used in Experiment XXVI., heated to 55° C.
4.40 Apparently no effect.
5.00 Injected 50 c.c. subcutaneously.
5.30 Temperature 39.8° C. Apparently not affected in any way.

Exp. XXVIII.—Dog, weight 17 pounds.
1.20 P.M. Playful, normal temperature 38.7° C.
1.30 Injected subcutaneously 75 c.c. blood serum from a case of uræmia.
1.45 Purged; seems sick; not playful.
2.00 Drowsy.
4.00 Still seems sick; temperature 38.8° C.

Exp. XXIX.—Dog, weight 20 pounds.
2.00 P.M. Normal temperature 38.6° C.
2.20 Injected 100 c.c. same serum as in Experiment XXVII., heated to 55° C. for ten minutes.
3.00 Playful, and does not seem sick.
4.00 Temperature 39.2° C. As playful as before the injection. Does not seem to be at all affected.

The dogs were placed as nearly as possible under identical conditions of environment and the injections done in the same manner, so that any disturbing element other than the injected substances would act in a similar manner in every experiment.

Human urine, both healthy and that from Bright's disease, urea, kreatin, and kreatinin produced uniformly a fall of temperature. As this was the most marked in those cases where large doses were used, it is probable that some of the fall, or all of it, may be due to the water injected, and not to any specific action of the solids.

In analyzing the effects of the injection of dog's urine it will be seen that when small quantities were used the same fall of temperature

resulted as in the other experiments, but when large quantities were injected the temperature rose. It is true that at first there was a fall, and then a rise occurred secondarily, at a period when observation had ceased in the other experiments, so that this fact may be for comparison robbed of a certain amount of value. It may be noted that in Experiment V., in which the renal arteries were tied, there was no such rise, so that it was not the result of the operation *per se*.

The experiments in which blood serum was used are not altogether definite in their results. However, it seems probable that uræmic blood contains a poison which is capable of producing a rise of temperature.

(To be continued.)

ROUND-CELLED SARCOMA OF THE ANTERIOR MEDIASTINUM:

Extensive Metastasis, including the Brain, Both Choroid Coats, Oculo-Motor and Optic Nerves, and External Ocular Muscles.

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An Italian laborer, aged twenty-one years, was admitted to the Pennsylvania Hospital October 1, 1888. The history is as follows: He was well and strong until June, when he had ague of the tertian type. For forty days before admission he was unable to work, owing to pain in the head and shortness of breath, which for ten days amounted to orthopnœa, and his breathing was wheezing. The urine was scanty and passed infrequently. On swallowing he had a burning sensation behind the sternum, and the act provoked coughing, but he did not vomit. He had no appetite and was wakeful at night. There was diplopia for at least ten days before coming to the hospital.

On admission the patient is a fine, strongly developed man. The face is dusky red and congested, the neck broad and very thick-looking, and its veins and those in the upper part of the chest full and turgid. The eyeballs are markedly staring, and there is slight external strabismus of the right eye. The tongue, when protruded, is not deflected to either side.

The arms and hands are cyanotic, but there is no cyanosis of the feet or legs. The dyspnœa is very urgent, and he is entirely unable to lie down. There is, perhaps, some slight difference in the volume of the two radial pulses at the wrists. The heart sounds are clear and no murmur is audible. There is dulness on percussion at the lower portions of the lungs posteriorly, and whistling, dry râles are to be heard over

the lungs everywhere. There is a roaring sound as if from obstruction of the trachea or bronchi, and this is more marked upon the right than upon the left side. He expectorates a good deal of watery, blood-stained fluid.

Death occurred in great agony from suffocation about twenty-four hours after his admission to the hospital.

The *post mortem* examination revealed the presence of a large fleshy mass lying behind the sternum. This was wedge-shaped, with the base above, and was perhaps three inches wide by five vertically. It lay behind the sternum and over the pericardium, and completely surrounded the bloodvessels and the trachea, except a little of its posterior wall. The trachea was much compressed and pushed or dragged to one side, being thus out of the median line.

The innominate artery divided within the mass, and its two branches were narrowed. The carotid, a short distance from its origin, showed destructive erosion so great that the wall was very thin. The left innominate vein was included within the tumor, and it and the carotid and subclavian veins which formed it were all markedly narrowed.

The heart appeared to be of about the normal size, and its valves and cavities and the openings looked natural. Upon the surface of the heart the vessels appeared tortuous and thick, and stood out in an unusual way. On cutting across such vessels they appeared to be surrounded with neoplastic material. At the apex there were several spots which were dark-red and looked like ecchymoses. At the base of the heart there was neoplastic material deposited upon the great vessels and on the auricles, and at one place this extended across to the parietal layer of the pericardium, which was ragged-looking.

The pleural sacs both contained bloody serum, the right being tightly distended, and the right lung compressed and carnified. Both lungs showed here and there upon their surfaces spots of neoplastic deposit, and upon the pleural surface of the diaphragm, upon the right side, there were a number of quite large nodules.

The liver was large, weighed four pounds fourteen ounces, and was very full of blood. On section it presented the nutmeg appearance.

The spleen was about five times the natural size and very firm in texture.

The mesenteric glands were greatly enlarged and were sarcomatous. The kidneys presented numerous nodules upon their surfaces, and on section areas of sarcoma were seen scattered through the parenchyma.

The brain itself presented no gross evidence of disease. The oculo-motor nerves were both enlarged, the right one being fully twice as large as the left, and presenting a pear shaped swelling just within the cranial cavity, before its entrance into the sphenoidal fissure.

Sections were prepared for microscopical examination of the primary growth which lay in the mediastinum. Of this, sections from three positions were cut, one taken from the body of the mass, and two including the right carotid artery—one of the eroded portion of the vessel, and the other of a portion in which the walls were still whole. Of the heart, sections were cut from two positions, one near the apex, and the other of the septum on the anterior surface, including an artery in the pericardium. Two sections of the lung were prepared from different positions. Sections were cut of mesenteric glands, of the diaphragm, including a sessile growth upon its pleural surface, of the liver, the spleen, and two from different parts of the kidney.

Of the nervous system, sections were prepared of the cerebellum, of both oculo-motor and of both optic nerves, just within the cranial cavity and before their entrance into the sphenoidal fissures and optic foramina—of the posterior portions of both eyes showing the nerve entrances, and of the optic nerves and surrounding muscles and nerves behind the eyeballs.

The total number, therefore, of different portions of tissue subjected to microscopical examination was twenty-two, and all of them exhibited, to a greater or less extent, sarcomatous infiltration. The infiltrating material was of the commonly described round-cell variety, and in sections stained with carmine the cells seized the pigment with avidity, producing a very intensely red color. This seems to be a marked peculiarity of the cells of sarcoma, the color generally being more intense even than that of carcinomatous growths.

The primary growth in the mediastinum, which very probably originated in some remnant of the thymus gland, presented the usual appearances of round-celled sarcoma. The only specially noticeable feature of the growth was the erosion of the carotid artery. The only portion of the walls of the vessel remaining upon the side where the erosion had occurred was the external fibrous coat; the muscular layer and intima had disappeared entirely.

The sections of the heart showed the neoplastic material to lie more abundantly in the adipose tissue upon the surface than anywhere else. It followed the lines of the connective tissue, the rings constituting the boundaries of the fat cells being infiltrated. In places where the amount of deposit was still moderate the centres of the fat cells retained their usual appearance of being empty, but where it was great the cells had pressed inward until the spaces were filled up, and the appearance was one of a solid mass of the neoplastic round cells. At the point of junction of the overlying fat with the muscular substance of the heart, the great abundance of the neoplastic material ceased abruptly, as though it had found the invasion of the muscular territory more difficult. The sarcoma cells, however, were plainly to be seen amongst the muscular fibres, especially along the connective-tissue lines, but they were few and sparse as compared with the vast numbers of them in the fat layer. The invasion by the sarcomatous cells had penetrated very deeply into the muscular tissue, following the lines of the connective tissue and lying between the fibres.

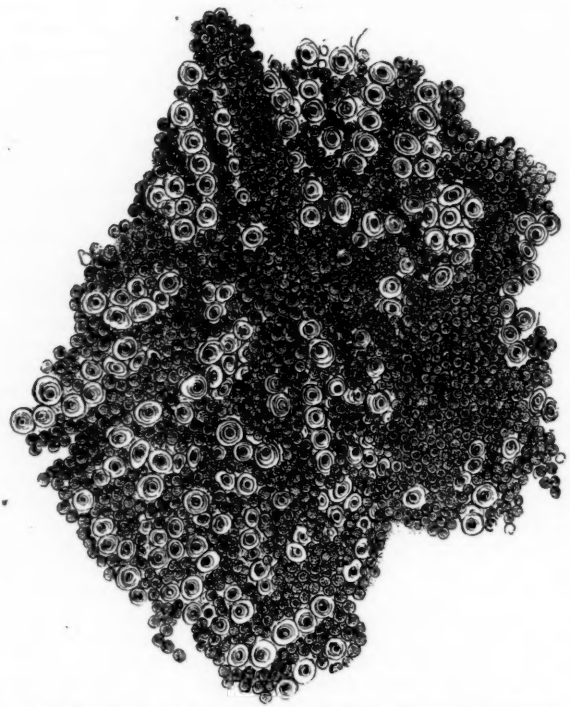
In the lung the sarcomatous infiltration had followed the connective-tissue lines, the pulmonary trabeculæ being enormously thickened and presenting themselves as masses of round cells. The walls of the air cells were also infiltrated in places, and where the growth had fully taken possession of an area, the cells had multiplied so that the air spaces were filled up, causing the lung to be quite solid. The nodules upon the pleural surface of the diaphragm were composed of masses of round cells, presenting no unusual features.

In the liver were many areas of varying size infiltrated with round cells, which had stained intensely red. None of these were large, nor had the macroscopical examination revealed the presence of any nodules sufficiently large to be seen with the naked eye. It seems fair, however, in view of the fact that sarcomatous infiltration had occurred so extensively into so many tissues and organs, to assert that these small

infiltrations into the liver were but an early stage of the same malignant disease which had progressed so much further in other places.

The spleen and mesenteric glands were sarcomatous, as was easily seen from their gross appearance, but as their natural histological appearances so closely resemble those of a round-celled sarcoma, the only especially noticeable characteristic of them was the intense red staining of their cells.

FIG. 1.



Section of the right oculo-motor nerve, showing extensive sarcoma infiltration.

The kidneys presented numerous nodules, both upon the surface and more deeply seated within their substance. These, when examined microscopically, were seen to be sarcomatous, the neoplastic cells lying between the bloodvessels and renal tubules in the connective tissue, except where the deposit had become very extensive, in which case the renal tissue was overwhelmed, and the appearance was simply that of a mass of the characteristic round cells. In one place there was found a layer, somewhat less than a thirty-second of an inch in depth, of sarcomatous deposit upon the surface of the kidney. The neoplasm included the capsule, and the separation of the diseased from the healthy renal

tissue beneath it was very sharply marked. Hemorrhage into this neoplastic material directly beneath the capsule had occurred.

The appearances of the sections of the cerebellum were not easy to interpret. The portions of pia mater included were infiltrated, especially around the bloodvessels, with cells precisely similar in appearance to those which in other places were beyond doubt sarcomatous. In view of this fact it seems fair to assert that the disease had invaded the pia mater, and this is the more just and reasonable, as a precisely parallel condition existed in connection with the eyes, as may be seen from the following description.

Left oculo-motor nerve. The individual nerve fibres are of normal appearance, axis-cylinder and medullary substance presenting their natural characteristics. Here the normal constitution ceases, and the nerve bundles and in many places the individual fibres are separated by collections of round sarcoma cells, which have closely followed the connective tissue septa and the bloodvessels supported by them. In other words, instead of the ordinary membranous investment of the fasciculi, this has been changed into one composed of the cellular elements of the sarcoma.

In the *right oculo-motor nerve* precisely the same process is repeated, only the infiltration of the perineurium and endoneurium is so extensive that the appearance is that of a section of ordinary round-celled sarcoma, through which are interspersed here and there single, normally constituted nerve fibres. (Fig. 1.)

Portion of the posterior half of the right eyeball and 9 *mm. of the optic nerve in longitudinal section.* Beginning from the extra-ocular end of the optic nerve and passing to the lamina cribrosa, there is no marked change in the nerve bundles and no increase in the nuclei. The trabeculæ of connective tissue are more developed than in the normal nerve, but they are not infiltrated with sarcoma cells. Toward the extra-ocular end of the nerve are several capillary vessels, around which are clustered numerous round cells. The lumen of the central arteries contains a few blood cells, and in addition small collections of larger, deeply stained lymphoid cells—a condition which repeats itself more decidedly in several adjoining vessels or branches. The outer sheath is thickened, but not infiltrated; the inner sheath is unchanged. The intervaginal space contains numerous darkly stained round cells gathered in the meshes of loose fibres. The nerve entrance is free from changes, evidences of neuritis being entirely absent.

The *retina* is badly broken and misplaced, rendering an accurate study of its elements difficult, which, save for some proliferation of the internal nuclear layer and distinct thickening of the fibre layer, are free from pathological changes. The pigmented epithelium appears only in fragments clinging to the choroid coat.

The *choroid* to the left of the nerve entrance, so far as the lamina vitrea and chorio-capillaris are concerned, presents no abnormalities. The bloodvessels of the stroma are well filled with corpuscles and some darkly stained cells. Here and there the leucocytes of this region are gathered together in small collections. The pigment cells, very dark, appear in characteristic variety of shapes.

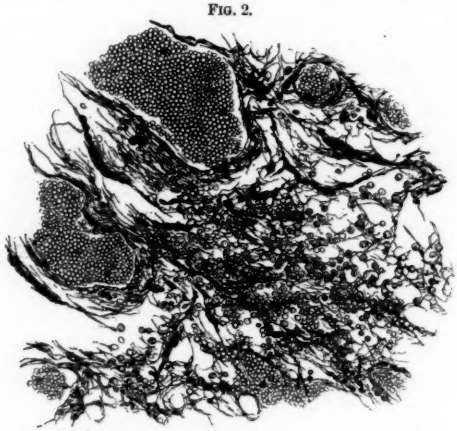
To the right of the optic nerve the choroid is detached from the sclera, thicker than on the opposite side, and, in addition to the appearances just described, presents in several spots in close relationship with vessels

in the stroma considerable collections of round, darkly stained cells, which, in general character and grouping, undoubtedly belong to the sarcoma infiltration.

The *sclera* is free from abnormal appearances, but in the branches of the ciliary nerves passing through it there are small round cells suggestive of those which have infiltrated the oculo-motor to such a great extent.

Posterior half of the left eyeball and 8 *mm. of the optic nerve in longitudinal section.* So far as the optic nerve, optic-nerve entrance, retina, one-half (left) of choroid and sclera are concerned, the description just given applies so nearly that it need not be repeated. The right half of the choroid remains *in situ* for 3 mm. from the optic-nerve entrance. Then it becomes detached from the sclera and is greatly thickened.

FIG. 2.



Section of the left choroid, showing large bloodvessels, pigmented cells, and secondary sarcoma deposit.

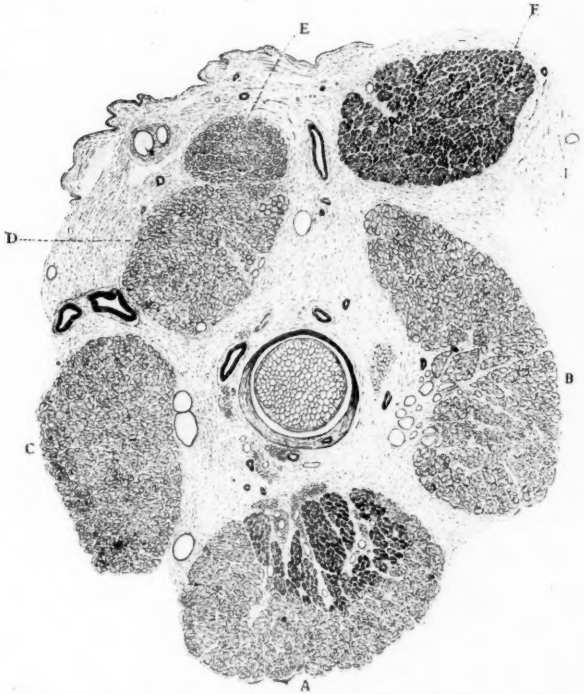
The branching, pigmented cells are massed together on the outer part, and also surround the well-filled vessels of the stroma, between which are gathered areas of round sarcoma cells, interspersed with pigment granules and dark, spindle-shaped cells. The chorio-capillaris can be traced unbroken throughout the section, but is indistinct directly over the thickest part of the affected choroidal tissue. The sarcoma cells are dispersed through the layer of choroidal stroma containing large bloodvessels, but are especially massed in several localized areas. Some of the larger veins contain darkly stained cells analogous in their appearance to those which lie outside of the vessel walls. (Fig. 2.) This area of infiltration begins 3 mm. from the nerve entrance, and is 1 mm. in diameter at its thickest portion.

Transverse section of the contents of the left orbit about 14 *mm. anterior to the foramen opticum.*[1] The relation of the parts (muscles, vessels, nerves, etc.) one to the other is undisturbed.

[1] This section corresponds almost exactly with Table VI. of Lange's Topographische Anatomie des menschlichen Orbital-Inhalts, and has been studied with the aid of this diagram.

Muscles. The external, internal, superior rectus, and levator palpebræ superioris are normal in appearance; the inferior oblique is not included in the section. The inferior rectus contains a patch, constituting about one-third of the section, situated in the upper part, which presents an

FIG. 3.



Transverse section of contents of the left orbit 14 mm. anterior to foramen opticum: A. Inferior rectus, showing patch of sarcoma in muscle. B, C, D, E. External, internal, and superior rectus, and levator, unaffected. F. Superior oblique; sarcoma between the optic nerve and the inferior rectus.

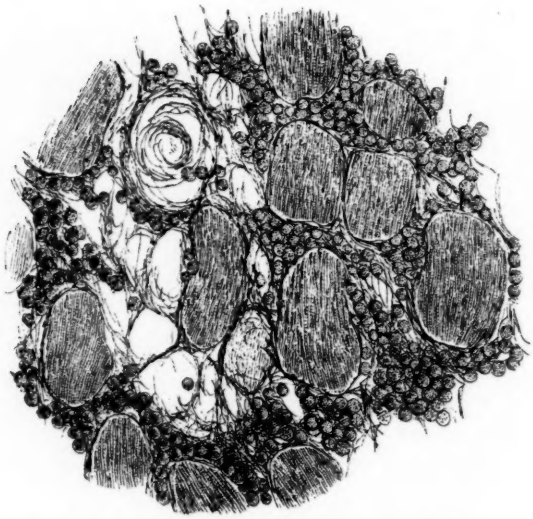
exquisite picture of infiltration with round sarcoma cells. The connective-tissue investments of the individual fibres and of the fasciculi are literally replaced with the cells of the new growth; while the sheath of areolar tissue covering the entire muscle in the affected area is crowded with the elements of the neoplasm, which extend into the surrounding fibro-fatty tissue of the orbit. For the most part the diseased area is sharply separated from the portions of the muscle which remain unaffected, although in places small trains of cells proceed a short distance into neighboring areas along the internal perimysium.

The section of the superior oblique is more freely supplied with darkly stained nuclei and corpuscles than those of the other muscles, and in one

or two spots, in the neighborhood of small bloodvessels, are collections of larger lymphoid cells, probably representing small foci of sarcomatous infiltration. (Figs. 3 and 4.)

Optic nerve. The nerve itself is normal, but in the intervaginal space, clinging closely to the inner sheath, are many small round cells, while in the fibro-fatty tissue, between the nerve sheaths and the inferior rectus

FIG. 4.



Section of inferior rectus, showing infiltration of sarcoma cells between fibres.

muscles are numerous collections of sarcoma cells, particularly immediately in relation with the cross-sections of the ciliary arteries.

Other nerves. The ciliary nerves, naso ciliary nerve, branches of the oculo-motor, supra-orbital, and a branch of the trochlear have been identified. In none of them are the appearances repeated which have been described with the main stem of the oculo-motor. Of the nerves just named the supra orbital is more richly supplied with corpuscles and nuclei than the others, but there are no sarcoma cells between the nerve fibrils. The branches of the nerves supplying the inferior rectus, contained within the body of the muscle, although themselves not infiltrated, are literally surrounded with sarcoma cells, which infiltrate their external connective-tissue investments.

Bloodvessels. The following bloodvessels are evident in the section: the supra orbital, ophthalmic, and ciliary arteries and branches of the ophthalmic veins. The coats of these vessels are free from infiltration, except in the case of the ciliary branches lying between the optic nerve and the patch of infiltration in the inferior rectus, and even in these the sarcoma cells lie rather in the loose fibro fatty tissue adjacent to the vessels than in the coats of the artery itself, although in some instances small foci may be seen in the adventitious tunic. In the small vessels

supplying the inferior rectus muscle this character of infiltration (*i. e.*, within the adventitious coat) is more conspicuous.

Transverse section of a portion of the contents of the right orbit. As the contents were fragmentary, the relation of the various constituents to each other, and consequently their identification, is imperfect.

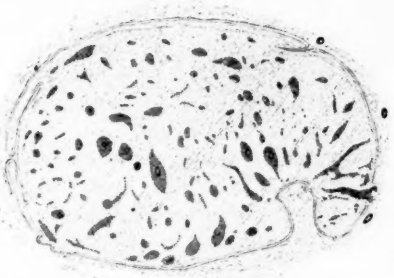
Muscles. The following muscles appear to be included: superior rectus, levator palpebræ superioris, a fragment of the superior oblique, and the internal (or inferior) rectus. These structures have escaped the sarcomatous infiltration, with the exception of the last-named muscle, which contains two patches of diseased tissue exactly similar to the one described in the inferior rectus of the left orbit. The branches of the oculo-motor nerve supplying this muscle are not only surrounded by sarcoma cells, but the individual fibres have been separated by the elements of the new growth precisely as the same process has occurred in the main stems of both third nerves.

The optic nerve is normal in appearance, but in the intervaginal space the collections of round cells are more evident than in the corresponding situation on the opposite side.

The bloodvessels, except the small branches which supply the muscle in the area of infiltration, are free from sarcomatous deposits.

Cross-sections of the optic nerves just within the skull. The dural sheaths are not visible in the section; the inner (pial) sheath is densely infiltrated with sarcoma cells, which, passing inward from this situation along the

FIG. 5.

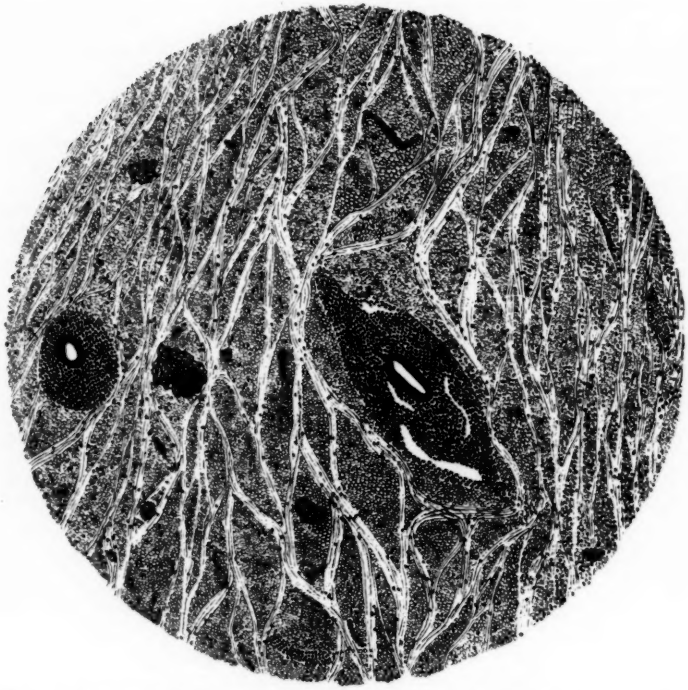


Transverse section of optic nerve just within skull, showing general plan of the sarcoma infiltration.

trabeculæ, infiltrate those in and within the margins of the nerves. In general, the nervous tissue appears normal, and there is no undue development of nuclei. Each capillary and arteriole, however, is surrounded with a considerable area of neoplastic cells, which completely replace the adventitious coat and perivascular sheaths of the larger vessels, and entirely fill the spaces surrounding the smaller ones. Within the lumen of many of the vessels are cells exactly the counterpart of those which surround them. (Figs. 5 and 6.)

The case seems worthy of being described and recorded, as sarcoma of the mediastinum is by no means common, and as the metastatic growths were more fully examined and studied with the microscope than can be or

FIG. 6.



Portion of Fig. 5 more highly magnified, showing arrangement of sarcoma cells surrounding the bloodvessels.

has been done in most of the cases recorded in the journals, but especially on account of the condition of the eyes and their appendages. As has already been said in connection with the description of the macroscopic conditions, as noticed at the post-mortem examination, nothing abnormal was seen except the difference in size of the two oculo-motor nerves. It would probably have escaped notice that the left, the smaller of the two nerves, was much larger than usual, had it not been for the fact that the right was still larger. The difference in size having been noticed, of course further investigation was made to discover the cause.

Since seeing this case, now four years ago, one of us (Dr. Meigs) has seen another, which was in many respects parallel:

A man, aged forty-three years, who had always been healthy, was for a month annoyed by slight cough; but it was so slight that it had not prevented his starting on a long pleasure journey. Two weeks later found he had shortness of breath, which soon became so severe that he was advised to come home. When seen he was dull of intellect and his mind disposed to wander; he had dyspnœa, and the face was much con-

gested and swollen. There was marked nystagmus, and the eyeballs seemed prominent. In the supra-clavicular fossa upon the right side was a tumor. This was not very hard and seemed to be pressing upward from the chest cavity, so that it almost entirely filled the fossa. It did not pulsate. Examination of the heart brought to light no evidence of disease, as the sounds were soft and clear, but the action was rather feeble. There were whistling and cooing râles to be heard over the lungs, but no signs of consolidation. The appearance of the lower extremities was in marked contrast to that of the head. The patient was a large, stout man, and the swollen head and bulging eyes hardly looked as if they could be a part of the same individual as the legs, which were emaciated and shrunken. The history was that the tumor had existed only two weeks. This, taken in consideration with the fact that there had been severe dyspnœa for the same length of time, indicated strongly the presence of a rapidly growing intra-thoracic mass. Such a growth could only be a sarcoma, and the opinion was therefore expressed that the disease was mediastinal sarcoma.

It seemed almost certain, too, in the light of the other case that has been described, that the staring of the eyeballs and nystagmus must be due to an invasion of some part of the ocular apparatus by metastatic sarcoma. Within three or four days a tumor showed itself in the other supra-clavicular space, the dyspnœa increased terribly, and he died. A post-mortem examination was made, and a sarcomatous tumor weighing one and a quarter pounds was found occupying the mediastinum. The growth surrounded the trachea, aorta, and other large bloodvessels, and there were secondary deposits ranging in size from that of a bean to a chestnut upon the diaphragm and posterior wall of the thorax. The post-mortem examination does not appear to have been very thoroughly made, for there is no mention of metastases to any of the organs, though such almost certainly must have existed, nor was any investigation of the brain or eyes made. It is much to be deplored that sections from the eyes and their appendages could not have been thoroughly examined with the microscope, for the disease had almost certainly invaded them. That which was observed in this case, imperfect as the investigation was, when taken in connection with the more fully described one, makes it desirable that metastases to the ocular apparatus in cases of mediastinal sarcoma should be sought for. It may be, too, that it occurs in cases of sarcoma arising in other situations than the mediastinum. The presence of the sarcomatous deposits in the ocular apparatus might readily be overlooked, for there are hardly any gross appearances to be seen. It is reasonable to suppose that there was more sarcomatous deposit somewhere in the brain than that in the pia mater of the cerebellum which has been described, and which could only have been discovered by careful microscopical examination.

As already indicated, a large measure of interest centres in the distribution of the sarcoma cells within the eyes, optic nerves, oculo-motor (third) nerves, and the orbital muscles. Both third nerves are exten-

sively infiltrated throughout their length, especially upon the right side. In the branches of the nerves in the left orbit no exactly similar appearance was found, except in those supplying the inferior rectus, which, while not themselves infiltrated, are densely surrounded by cells. Again, in the right orbit, of the muscles apparently identified, a portion of the internal (or inferior) rectus is contaminated, as also are the nerves supplying it, not merely in the form of surrounding areas of cells, but also by a separation of the individual fibres with the elements of the new growth, exactly as this occurs in the parent stem. Therefore, if the appearances seen in the left superior oblique are not accepted as certainly sarcomatous, we may infer that one pathway of the morbid process was along the oculo-motor nerves, and through it to two, possibly three, of the muscles supplied by them.

The optic nerves within the skull are extensively diseased, but the process of infiltration grows less marked as they approach the eyes themselves. Indeed, even in the cross-section in the orbit scarcely any diseased tissue is evident, while in the portions of these nerves examined in connection with the posterior halves of the eyeballs, save for some slight cellular infiltration around the capillary vessels and in the intravaginal space, evidence of sarcoma is lacking. Therefore it may be inferred that a second pathway of the morbid process was *via* the optic nerves, and a striking and interesting feature of the case at once becomes apparent, viz.: that the disease was more advanced and extensive in degree within the skull than in the orbital and ocular termination of the third and second nerves. In other words, this indicates that the disease travelled along these pathways from the brain forward toward the eyes.

The deposits of sarcoma cells in each choroid, but particularly in that of the left eye, give this case importance in the literature pertaining to sarcoma of the uveal tract. The fact that these deposits are limited to a certain area of the choroid, that they are surrounded by comparatively healthy tissue not contiguous with infiltrations elsewhere located, and that the bloodvessels within them and in their immediate vicinity contain cells apparently of the same character, affords evidence that they should be regarded as metastatic nodules, probably of embolic origin.

Fuchs, in his admirable monograph, *Das Sarcom des Uveal Tractus*, Wien, 1882, having analyzed the entire literature to date, states that metastatic choroidal sarcoma is unknown, and quotes Virchow's well-known sentence: "Those organs which exhibit a great tendency to protopathic tumor formation present a very slight inclination to metastatic deposits." Fuchs refers to, but rejects, Broemser's case[1] of supposed metastasis of a melanotic growth of the cheek to the choroid, for the excellent reason that no microscopic evidence is at hand that either the ocular or the facial growth was sarcomatous, and reiterates his belief

[1] Ueber einen Fall v. secundär Melanom. d. Choroid. Diss. Inaug., Berlin, 1870.

that up to the date of his writing sarcoma metastasis to the choroid had not been observed.

Pflüger,[1] of Bern, contributes a case of sarcoma in a young woman, aged thirty years, which developed from a congenital nævus in the region of the right parotid gland, and which was followed by secondary sarcoma of the glands, with metastasis into the skin of the back and head, into the right choroid, and probably into the cerebrum. Three years and a few months elapsed between the period when the congenital pigment-patch began to enlarge and the death of the patient. Autopsy was refused, and the facts reported are based upon clinical observation alone. Ophthalmoscopic examination revealed a tumor occupying the medial half of the eye-ground. Pflüger refers to another case of intra-ocular metastatic sarcoma reported by Schiess-Gemuseus,[2] the primary growth being a sarcoma originating from a congenital nævus in the parotid region. Ophthalmoscopic examination was impossible, owing to opacities in the media, but the microscope showed that the secondary growth had originated from the optic papilla. Hence the case is not properly classed with choroidal growths.

In the case forming the basis of this paper unfortunately ophthalmoscopic examination is wanting, but the anatomical studies are sufficiently detailed to demonstrate secondary choroidal sarcoma. It is scarcely conceivable that the choroidal growth under these circumstances could have been the primary one, although, as is well known, in a few instances extremely small and totally unsuspected growths in this situation have been followed by very extensive metastasis; in one case, quoted by Fuchs, a melanotic mass was found in the heart secondary to a small sarcoma in a sunken eyeball, which had existed in this condition for more than twenty years.

Independently of the fact that the sarcomatous deposits in the eye were confined within the scleral walls, and were both clinically and microscopically of the nature of secondary growths, the mediastinum rarely suffers from this form of tumor, save as a primary growth. Among ninety-eight cases reported by various authors and collected by H. A. Hare,[3] but five were secondary and thirty-one were primary, the remaining number having no distinct reference in regard to this point. The interesting fact has been noted that so far as the optic nerves, third nerves, and orbital contents are concerned, the microscopic evidence is that the disease travelled from the brain forward toward the eyes along these pathways, but the localized character of the choroidal neoplasms and their failure to be in any demonstrable connection with other sarcomatous areas, seems definitely to indicate that they should be regarded as embolic deposits.

[1] Archives of Ophthalmology, vol. xiv. p. 185. [2] Graefe's Archiv, Bd. xxv., Abth. 2.
[3] The Pathology, Clinical History, and Diagnosis of Affections of the Mediastinum. Fothergillian Prize Essay. Philadelphia, 1889.

REVIEWS.

THE YEAR BOOK OF TREATMENT FOR 1894. A Critical Review for Practitioners of Medicine and Surgery. By Twenty-four Contributors. Pp. viii, 492. Philadelphia: Lea Brothers & Co., 1894.

WHEN one reads this work he cannot but be impressed by the marked and substantial advances, which have been made in both medicine and surgery. To faithfully record these advances and in the selection of the material to discard the hasty generalizations upon insufficient data, to eliminate the optimistic conclusions of inexperienced observers, to make due allowance for dogmatism based upon preconceived notions, is by no means an easy task when one considers the enormous number of papers which are assumed to be contributions to medical literature. The practitioner will find many abstracts of articles which have been noticed in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES in the department of Progress, and with which he is already familiar, yet the work will have none the less of interest to him, for the time which has elapsed since their publication has brought them into a truer perspective, although, of necessity, the *résumé* cannot be brought down to so nearly the time of this writing. That the *Year-book* has filled a need of the profession is clear, for this is the tenth volume of the series, and the end of its first decade is noteworthy in that two new departments have been added—those of the Medical Diseases of Children and of Bacteriology. When the work has been so well done, the selection so careful, the editorial comments so fair, although at times incisive, it might appear ungracious to point out departments of especial merit. We believe, however, that the work of Dr. Dudley W. Buxton on "Anæsthetics" should be carefully studied by those whose practice brings them in contact with anæsthesia. Dr. William Hunter, in his review of "Bacteriology" in relation to treatment, gives an excellent *résumé* of the discoveries in the laboratory which have a practical bearing upon this new field of therapeutics—the tetanus antitoxins and the serum-therapy. The summary of the therapeutics of the years 1892–93, chiefly with reference to new remedies, by Dr. Walter G. Smith, is somewhat disappointing in that the recent candidates should have the fullest possible information presented rather than a mere outline. To be sure, much of such information will soon be rendered superfluous by the failure of many remedies to meet the expectations of their purposes, but the fuller the knowledge the earlier will be demise of the useless drugs, and the sooner will literature cease to be encumbered with them. The selected list of new books, new editions, and translations does not include many works of importance which have appeared within the time specified; it is, however, a fair record. The painstaking labor which has been expended upon every page may not be apparent to the reader, but a careful study of this work will demonstrate its presence. We are

familiar with the various year-books, and while our judgment as to which are the valuable contributions to medical knowledge does not always coincide with that of the editors, we are firmly convinced that every practitioner should review his mental storehouse, should square his information with the general verdict of the profession, and thus remedy his deficiencies. Of all the yearly summaries with which we are acquainted we believe this to be the best, and we base this opinion upon the care shown in its compilation, the condensation of statement, the infrequent instances of repetition, the breadth of the field which is covered, the editorial comments, which are critical in the true sense of the word, and, lastly, but by no means of the least importance, the very excellent mechanical make-up. R. W. W.

CHARAKA-SAMHITA. Translated into English. Published by AVINASH CHANDRA KAVIRATNA, Editor of Susruta-Samhita, with commentaries; Bengali and Hindi Translator of Charaka-Samhita, and of Susruta-Samhita; Editor of Chikitsa-Sammitani, etc. Parts IV.–VIII., pp. 89–248. Calcutta: D. C. Dass & Co., 1892.

ONE cannot read this translation of ancient medical lore but he must admit the close study, the logical deductions, and the concise generalizations of the sages of antiquity. When we consider the disadvantages under which they labored—chemistry and physiology yet unborn, anatomy almost an unopened book—crude practice must of necessity prevail. Botany was further advanced than pharmacy, but even that science was primitive as we understand it to-day. The first three parts, reviewed in an earlier number of THE JOURNAL, contained a list of remedies, many easily recognized as in use to-day. The fourth part commences with an enumeration of hygienic rules for the person and general rules of conduct, many of which are quaint, some deserve to be handed down as epigrams, and others appear to be trivial; all, however, possess a great interest for the scholar. In the succeeding parts the remedies, as applied to special diseases and conditions, are fully set forth, and one cannot be but impressed by the evidence of keen observation which is to be found on every page.

The lesson devoted to some of the diseases of the head is so full in the statement of the disease-causes that we scarcely imagine that we are reading a work more than twelve hundred years old. So, again, in the lesson upon swellings and inflammations, we find the important conditions clearly set forth. In the eighth part we find a classification of the diseases that usually occur—the accidental and constitutional; the relation of diseases depending upon their producing pain, their substrata, mind and body; their causes, and the kinds of drugs and operations necessary for treatment. The censurable faults of man physically are enumerated—excessive tallness, shortness, hairiness, baldness, darkness and lightness of complexion, obesity and leanness, the last two being of especial moment. The proper medicine for the particularly censurable ones is also indicated. This part also contains some sage observations upon sleep, together with the remedies for excessive and deficient sleep. The part finally closes with the six principal forms of treatment:

1. Langhana, that which lightens or attenuates the humors of the body.
2. Vringhana, that which promotes the nutrition and bulk of the body.
3. Rukshana, that which brings about dryness, roughness, or paleness.
4. Snehana, that which leads to secretion of oily matter, softness of the body, and increase of impurities.
5. Swedana, that which produces perspiration and destroys stiffness and heaviness and sensation of cold.
6. Stambhana, that which constipates or stops the motion of such constituents of the body as are liquid, restless, and endued with motion.

These various treatments are thoroughly explained so that they can be understood. The translator is placing under obligation the entire medical world in presenting this excellent translation. Those of the younger generation of physicians are too likely to esteem as of little importance the learning and diagnostic acumen of the sages. A work like this is important not only for its showing our indebtedness to the fathers of medicine, but as well it is likely to be the starting-point of new discoveries, whose suggestion lies in the pregnant aphorisms and proverbial sentences of this work. We await the succeeding parts with interest.

R. W. W.

TREPHINING IN ITS ANCIENT AND MODERN ASPECTS. By JOHN FLETCHER HORNE, M.D. 12mo., pp. xii., 133. London: John Bale & Sons, 1894.

DR. HORNE has written a very excellent brochure in six chapters, which take up first the early literature of trephining and its modern application to diseases and injuries of the head and spinal cord, and then a brief summary in the succeeding chapters of the application of trephining to intra-cranial hemorrhage, abscess, cyst, tumor, epilepsy, paralysis, psychoses, and gunshot wounds of the head. A short chapter is given to trephining the spine, and the book closes with some general remarks on the technique of trephining. He shows that he has had, under Mr. Wheelhouse's excellent tuition at the Leeds General Infirmary, not only a practical training in surgery, but also a general acquaintance with the literature of this subject. There is but little in the book that is new to anyone who is familiar with recent cerebral surgery, but there are many to whom these pages will be a surprise, especially when they contrast the early literature of trephining as shown here, even down to the days of Bell and Petit, with our present methods.

In his introduction he makes a very pleasing allusion to his trans-Atlantic brethren, and says: "No apology is needed for the frequent references I have made to American surgery; I cannot but admire the genius and assiduity of the surgeons of that country, nor can we grudge them a large share of the honor for the great advances made in the department of brain surgery." We could wish, however, that he had always got the names of his American friends correctly. Thus, on page 104, the late Prof. S. W. Gross appears as "Gosse." On page 8, Detmold, of New York, is "Dutmold." In the footnote on page 78, Dr. Lanphear appears as "Lamphear;" while on the succeeding page, although he gives no reference to the writer mentioned, we suppose that "Kocker" is a misprint for Kocher.

There are introduced into the book a few cases which are of much interest, especially one on page 63, in which a man received a blow on the head with a sharp-pointed piece of iron on May 13th, walked three miles to his home, and had the bone removed on July 20th, nearly ten weeks after the injury, with complete recovery. W. W. K.

CLINIQUE DES MALADIES DU SYSTÈME NERVEUX—M. LE PROFESSEUR CHARCOT. Leçons du Professeur, Mémoires, Notes et Observations pendant les Années 1889-90 et 1890-91, et publiés sous la direction de Georges Guinon, Chef de Clinique, etc. Tome II. Paris, 1893.

A SPECIAL and melancholy interest attaches now to any publication bearing the name of Charcot. It is but a few months since the death of the illustrious savant whose name is identified with that of the Hospital of La Salpêtrière, and whose publications, in such profusion, have at short intervals carried the fame of French neurology abroad.

The book before us is a continuation of one of those series of clinical studies which we had come to expect with regularity from Charcot and his assistants. Its predecessor was reviewed in these columns, and was notable for some very able papers in Charcot's best style. The present volume is given over rather too much to the collaboration of the great clinician's assistants, who have received an indulgence in space and opportunity which marks the generosity of the master toward those who have served him. This attitude in Charcot was always conspicuous, and to us in America, who know but little of the relations of master and assistant, was always rather striking. Not a little of his fame will rest upon the work that he inspired others to do; and it must always remain a proof of his strong personality and of his ability to impress this upon the minds of his disciples that much of their literary work has been received and applauded because the master himself shines through all their pages, and has almost invariably ennobled each of their books with the dignity of a preface.

On the whole, we do not find this volume quite so attractive and useful as was its predecessor. This may be due to the fact that we find fewer lectures by Charcot and more work by his assistants. In truth, the book is largely the work of Guinon and Sophie Woltke. The former has no reason to apologize for any of his excellent literary work, either here or elsewhere; but he is bound, perhaps, to suffer a little neglect when he publishes his papers under the same covers with Charcot. This is one of the penalties of associating with greatness. In the present volume there are but five lectures by Charcot.

The first of these is an elaborate study of a case of simple focal epilepsy caused by a circumscribed tubercular meningitis in the paracentral lobule in a case of advanced pulmonary tuberculosis. The author claims that this localization is in the adult a very common one for tubercular meningitis (in contrast with the basal meningitis of children), and that this fact is explained by the distribution of the bloodvessels in the part, which distribution conduces to great richness of supply with sluggishness of blood-currents. The paper is notable for its orderly arrangement of facts, for its fulness of details, for its clear intelligence, and for

its almost complete ignoring of the great work in localization done outside of France.

The one other of these lectures by Charcot himself that seems to us to be especially worthy of mention is the last, on hysterical hemianæsthesia and toxic hemianæsthesia. The author propounds two queries: first, Is there a saturnine hemianæsthesia arising directly from chronic lead-poisoning? and, second, Is there an alcoholic hemianæsthesia belonging properly to the nerve accidents of chronic alcoholism? As is well known, Charcot and his school answer these questions in the negative. The hemianæsthesia, both sensorial and sensitive, which is observed occasionally in cases both of lead and alcoholic poisoning, is not toxic but hysteric. The present lecture by Charcot is devoted to demonstrating this fact clinically, and hence to justifying this nosography. The patient, a young house-painter, had hystero-epilepsy, with hemiparesis, contractures and hemianæsthesia. He had also the gingival blue line, skin-discoloration and anæmia, but no history of wrist-drop, lead-colic, or arthralgia. Charcot differentiates the nerve-phenomena from lead-encephalopathy and from epilepsy, and proves their identity with hysterical manifestations. The lecture is the most important thing in the present volume. The French, almost exclusively, have recognized the toxic origin of hysteria, the symptoms of which, when observed in alcoholic and plumbic cases, have been confounded almost universally elsewhere with the effects of organic poisoning. This lecture will do much to make the subject clear to those who will take the pains to read it.

A valuable paper in this series is a review by Blocq, based upon lectures by Charcot, of the several forms of somnambulism. It has been apparent for some time that confusion exists in the use of the term somnambulism. Those who have followed the recent progress of studies in hysteria and hypnotism must have noted the frequent use of the term for describing a not uncommon stage of these disorders. It has been evident that the somnambulistic state of hysteria and hypnotism is something radically different from the ordinary somnambulism, or "noctambulism," which is seen occasionally as a disorder of sleep in otherwise healthy persons, and to which the term formerly was confined. Again, it has been recalled that a somnambuloid state is seen occasionally in epileptics, either as a substitute for, or as a sequel to, a fit. The necessity for a proper differentiation of these several forms has been evident, and in this paper M. Blocq meets the demand. Somnambulism is divided into two classes, the natural (noctambulism) and the pathological. The latter is again subdivided into epileptic and hysteric, which latter includes the hypnotic variety. The only fault we find with the paper is that the author finds it necessary at all to group these diverse states in one category. They are essentially distinct, and they are brought together only because they have been confused by a misuse of terms, especially in France. The somnambulism of healthy child-sleep is a widely different psychical state from the somnambuloid state of hypnotism, and this again from the automatism of epilepsy. There is no reason for grouping them together; all that is needed is the abolition of the term somnambulism as applied to any state either of hysteria or of epilepsy.

Guinon also furnishes in this book some valuable contributions to this subject of so called hysterical somnambulism. Then comes a lecture by Charcot on "Vigilambulism Hystérique," a term proposed by Egger

and Lereboullet for the *second state* in cases of double personality. This lecture by Charcot is an interesting discourse on this subject as illustrated by the history of a patient.

The book, perhaps, is devoted rather too largely to hysteria and hypnotism to suit the taste of some readers. When, however, we recall the services done by the French school in these studies, and the fact that Charcot is now no more, we cannot but read with interest and profit.

Among other valuable papers are one on the "Association of Tabes and Diabetes Mellitus," by Guinon and Souques, and another on "Conjugate Paralysis of the Sixth Pair," by Blocq and Guinon. J. H. L.

SYSTEM OF DISEASES OF THE EAR, NOSE AND THROAT. Edited by CHARLES H. BURNETT, A.M., M.D., Emeritus Professor of Otology in the Philadelphia Polyclinic; Clinical Professor of Otology in the Woman's Medical College of Pennsylvania; Aural Surgeon to the Presbyterian Hospital, etc. Vol. I., pp. 789. Philadelphia: J. B. Lippincott Co., 1893.

THE first volume of this "system" is divided into Part I., Diseases of the Ear, which embraces thirteen papers by different authors, and Part II., Diseases of the Nose and Naso-pharynx, which embraces ten individual papers.

The commendable purpose of the editor is to foster in theory and practice a more intimate association of these subjects—parts which bear to each other the closest anatomical and pathological relations.

The authors have been selected with much wisdom, and their essays are highly creditable, yet the *tout ensemble* is still suggestive of the dictum that aurists are prone to treat but incidentally the throat, and laryngologists but incidentally the ear, for in the distribution and assignment of topics the ear receives ideal and exhaustive consideration, while the department of the nose and naso-pharynx sometimes fails of that degree of completeness which one expects in a "system."

Dr. William S. Bryant describes admirably the "Anatomy and Physiology of the Ear," devoting nearly one-eighth of the entire volume to these subjects, and introducing sixty-five well-selected figures which assist in elucidating the somewhat intricate anatomy of this organ. The same author gives an excellent *résumé* of the various tests of hearing, and concludes that "after all, the voice and the watch are the tests one usually falls back upon."

Dr. Huntington Richards describes the various aural instruments, and the methods of using them, while Dr. Edward B. Dench writes on "Congenital Malformations and Diseases of the Auricle," following which is a chapter on "Otitis Externa," including a truly admirable article on "Otomycosis," by Dr. Robert Barclay. He describes no less than thirty-four parasitic fungi which are prone to dwell and establish inflammation in the external auditory canal, and appends a bibliographical synopsis which will prove invaluable to those who will endeavor further to identify and classify aural moulds.

Sir William Bartlett Dalby, in writing upon the subject of the removal of foreign bodies from the external auditory canal, says: "There are no difficulties worthy of the name, excepting so far as these are

created by the attempts at removal unwisely and unhappily made by those who are inexperienced in the examination of the ear." Dr. Gorham Bacon's contribution on "Diseases and Injuries of the Membrana Tympani" is illustrated by the recital of numerous cases, possibly tedious to some, but convincing to the many, and there are yet aurists who can profit by the same author's careful study of the etiological relationship of "adenoid vegetations" to acute otitis media.

Certainly no one more acceptable than Dr. Samuel Sexton could have been selected to present the subject of "Chronic Catarrh of the Middle Ear," for he represents a material advance in the treatment of this most persistent affection, that is, the operation of excision of the drum head and the two larger ossicles. The technique of this operation is briefly but plainly given, and is again described at length by Dr. Charles H. Burnett, the editor, in his personal article on "Chronic Purulent Otitis Media," with illustrations of the necessary instruments and of the electric forehead-lamp employed. Then follows the experience of Dr. Frederick L. Jack with the still more recent operation of removal of the stapes in chronic catarrhal and in chronic suppurative otitis media, with a description of sixteen cases in which uniformly good results were obtained so far as concerns the tests of hearing by ordinary and whispered voice.

Novel ideas may be gleaned from Dr. Christopher J. Colles' chapter, especially that part of it which relates to reflex relationships between the middle ear and dental, pharyngeal, and nasal affections. "Inflammation of the Mastoid Portion of the Temporal Bone," by Dr. Clarence J. Blake, scarcely receives as full and precise a treatment as the importance of the subject would seem to suggest; for instance, the operative measures for opening into the cranial cavity when necessary are accorded a space of less than one page.

Part II., Diseases of the Nose and Naso-pharynx, embraces chapters on "Anatomy" and "Physiology," by Dr. Alexander MacCoy; "Methods of Examination and Diagnosis," by Dr. Samuel G. Dabney; "Local Therapeutics," by Dr. Clinton Wagner; "Acute Rhinitis," by Dr. F. H. Bosworth, valuable for its plain statements concerning the proper regulation of the clothing and the use of the cold bath as prophylactics; "Influenza and 'American Grippe,'" by Dr. Carl Seiler; "Chronic Hypertrophic Rhinitis," by Dr. Ralph W. Seiss; "Cirrhotic (atrophic) Rhinitis," a masterly article, by Dr. John N. McKenzie; "Lupus, Tuberculosis, Syphilis, etc.," by Dr. George W. Major; "Foreign Bodies in the Nose and Epistaxis," by Dr. E. Cresswell Baber; and "Diseases of the Accessory Sinuses of the Nose," by Dr. Joseph H. Bryan—all admirable papers individually, but which, collectively, can hardly be accepted as composing a complete treatise on diseases of the nose and naso-pharynx. For instance, nasal polypus is disposed of in the space of eight lines under the title of "Hypertrophic Rhinitis," with an additional bare reference under "Disease of the Ethmoidal Sinuses," while nasal fibroma and the more common naso-pharyngeal fibroma are unmentioned. Again, the subjects of adenoid vegetations and of septal excrescence are but meagerly treated, and that within the scope of another title, while deviation of the septum narium also fails of any mention.

The volume presents an elegant appearance, being provided with good paper, clear type, and well executed illustrations. W. E. C.

PROGRESS

OF

MEDICAL SCIENCE.

THERAPEUTICS.

UNDER THE CHARGE OF

REYNOLD W. WILCOX, M.A., M.D., LL.D.,

PROFESSOR OF CLINICAL MEDICINE AND THERAPEUTICS AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL; VISITING PHYSICIAN TO ST. MARK'S HOSPITAL.

Bone-marrow in the Treatment of Pernicious Anæmia.

Dr. Thomas R. Fraser reports a single case where the curative effect was sufficiently evident to justify its publication. The patient was a man aged sixty years, suffering from frequent vomiting, diarrhœa, œdema pedum, moderate and irregular pyrexia, dyspnœa, and finally complete prostration. The hæmocytes varied from 1,860,000 to 1,460,000 per c.mm.; hæmoglobin 28 to 30 per cent. Ox bone-marrow was given by the mouth, uncooked, and in the daily quantity of three ounces. After twenty-seven days the hæmocytes rose to 3,900,000 per c.mm., the hæmoglobin to 78 per cent. The frequent failure of therapeutic measures in pernicious anæmia confers an interest upon any remedy which appears capable of controlling this malignant disease, even although the evidence is derived from one case only, and notwithstanding the fact that temporary improvement occasionally, though very rarely, appears to occur spontaneously. It is hoped that bone-marrow will be found to have a remedial value in some, at least, of these cases of pernicious anæmia.—*British Medical Journal*, 1894, No. 1744, p. 1172.

The Treatment of Sporadic Cretinism by Thyroid Juice.

Dr. George E. Anson reports a single case treated for one year. A girl, aged ten years, of the size of a child of five, of good intelligence, but presenting the typical symptoms of the disease, received each day one lobe of sheep's thyroid, freely minced and eaten raw. As this amount gave rise to feverishness and violent vomiting, the treatment was discontinued for a week, then resumed with a piece of the thyroid equalling the size of a haricot-bean. Later a glycerin extract was used, being freshly made each time. During the year she gained four inches in height, her hair became thick and abundant, her skin natural and healthy, and the face has lost almost all trace of its former condition.—*Lancet*, 1894, No. 3687, p. 1063.

Dr. Telford Smith reports an instance of a boy aged twelve years who received not quite a quarter of one lobe of fresh sheep's thyroid minced and mixed with some warm rice and jam at tea-time. On the following day headache and vomiting set in, and he was depressed, pale and cold. Six days after the treatment was recommenced, with one-eighth part of the fresh lobe, and continued for about two months, the improvement being marked. During two months without treatment he began to revert to his former condition. He was now given a tablet, which is equivalent to one-twelfth of a lobe, and this treatment has been followed by success. After five months the improvement is very marked.—*British Medical Journal*, 1894, No. 1744, p. 1178.

Dr. T. C. Railton reports the case of the older brother of the preceding. The treatment was commenced with thirty-six grains of raw thyroid, but as the first dose was followed by fever, vomiting, and restlessness, and the subsequent three by marked loss in weight, he was given two thyroid tablets of five grains each, and this number was gradually increased until the maximum of five was reached. The improvement in bodily growth and cerebral functions has been continuous; but time alone will determine to what point his mental development can be made to attain.—*British Medical Journal*, 1894, No. 1744, p. 1180.

The Action of Extracts of Muscles, Arterial Blood, and of Urine upon the Temperature.

M. H. Roger has carried out a carefully-conducted series of experiments, rabbits and dogs being used, from which he derives the following conclusions: 1. The extracts of the muscles contain thermogenic substances, while it is impossible to say whether these substances pre-exist in the tissues or originate during the manipulations which they undergo. 2. The entire arterial blood possesses a slight temperature-reducing property. 3. The entire arterial blood is sometimes thermogenic; this effect is noted when one takes it from a sick animal or exposes it to cold; in the latter case the thermogenic effect disappears when the animal is placed for twenty-four hours in a warm place, or when one has made a preliminary venesection. 4. Defibrinated blood, serum, and the exudates of pleurisy and of hydrocele produce elevations of temperature sometimes preceded by a slight lowering. 5. Urine causes a lowering of temperature, secondarily an elevation; this latter effect appears to be more marked when the subject which has furnished the urine has performed muscular exercise than when he has remained in repose.—*Archives de Physiologie*, 1894, No. 2, p. 216.

The Treatment of Chronic Intermittent Fever.

Dr. Spiridion Kanellis, during the past ten years, has treated five hundred and twenty cases of this disease, and has cured them. The patient takes four pills every morning at half-hour intervals, each containing six grains of quinine sulphate, one-fiftieth of a grain of sodium arsenate, made up with extract of quinquina. These pills are taken every morning for two weeks, interrupted for a week, then resumed for a second series, and so continued during three months. Also for the same length of time before the mid-day and evening meals, a coffee-cupful of an infusion of five grains of quinquina

bark and five grains of the herb absinthium in twenty ounces of water, to which has been added about a drachm of dry extract of quinquina and two ounces of cognac. Two hours after taking the pills the patient drinks a glass of milk. For the mid-day meal he has good soup, beefsteak, eggs, etc., with old wine. In the evening the same diet, and he is advised to go to bed as early as possible.—*Bulletin général de Thérapeutique*, 1894, 14e livr., p. 325.

The Use of Guaiacol for Neuralgia.

M. Ferrand reports that he has made use of the analgesic action of this drug by painting it, mixed with an equal part of glycerin, over the surface in sciatica and intercostal neuralgias. He has obtained relief of pain without lowering of temperature or phenomena of collapse. He has found that there is some cutaneous absorption, inasmuch as the drug has been found in the urine, but in small quantity.—*Journal des Praticiens*, 1894, No. 30, p. 359.

The Tubercular Diathesis Controlled by Gold and Manganese in Combination.

Dr. J. Blake White has made use of these remedies for several years. He administers the gold and sodium chloride with manganese iodide, in five minims of a one per cent. solution of carbolic acid, hypodermatically, daily or upon alternate days. A second solution has been made, consisting of the above-mentioned remedies in combination with strychnine arsenate and tincture of gelsemium. The somatic changes effected by these remedies are exhilaration, restoration of functional activity of both the nutritive and respiratory organs, increased appetite, corrected assimilation, manifest amelioration of important symptoms. It is not easy to explain the exact action of these remedies, but he believes that gold, by preference, exerts its effect upon the lymphatic and glandular systems, exciting them to healthy action, thereby disposing of unhealthy exudations and promoting assimilation, which is in time followed by marked gain in body-weight and decided improvement in general health.—*American Medico-Surgical Bulletin*, 1894, No. 7, p. 388.

Sommerbrodt's Creosote Capsules in the Treatment of Tuberculosis.

Dr. W. Thornton Parker believes that creosote is the best local remedy; it controls the cough, it is healing to the diseased tissues, and it vigorously attacks the seat of the disease itself. If persistently and scientifically used to its full extent, it penetrates the system so thoroughly that it becomes a resident antiseptic. The capsules of Sommerbrodt (creosote and cod-liver oil) are admirably suited to the creosote treatment. For hygiene: nutrition, attention to the condition of the mouth, stomach, and digestive systems, regularity of the bowels, of the meals, wholesome cooking, simple but nutritious; freedom from care and worry, a wholesome and abundant water supply, suitable clothing, exercise, well-ventilated sleeping-rooms, well protected from the winds, sunny, bright, and with an abundance of pure air; hygiene of the person, bathing, friction with towels, a comfortable bed—should be

included in the painstaking effort systematically made to overwhelm the tuberculous poison.—*Notes on New Remedies*, 1894, No. 11, p. 163.

The Antiseptic Treatment of Tuberculous Phthisis.

Dr. Arthur Foxwell states that there are four main ends to accomplish: 1. The prevention of the entrance of the bacilli of tubercle into our bodies or into the bodies of domestic animals. 2. The destruction of the bacilli which exist in the waste products of the disintegration of tissue induced by their action. 3. The destruction of the bacilli which exist in the still living tissues. 4. Nullifying the septicæmia which results from the ptomaïnes and various pyogenic cocci, which arise as secondary products of bacillary life. The first means scrupulous disinfection of dwellings and articles of personal use of tuberculous patients, the forbidding of their marriage, the exposition of the wickedness of ubiquitous spitting. To obtain the second end, complete sterilization of the sputum is necessary; also the sterilization of the excretion, before it leaves the body, by topical measures. The third end is the real one which we strive for in treatment. It may be attained by (*a*) endeavoring to make an antiseptic solution of the blood and lymph; (*b*) increase the cardiac strength so that there may be a more vigorous circulation through the bacillary districts; (*c*) enhance the respiratory interchange of gases, thus keeping the body as thoroughly oxygenated as possible; (*d*) increase the nutrition of the lung parenchyma; (*e*) increase the positive chemiotactic power of the bacilli, and also the capacity of the phagocytes for devouring bacilli. To nullify the septicæmia we must destroy the pyogenic cocci which cause it; hectic should be treated upon the same principles as we treat pyæmia arising from other causes. Inhalation is the feeblest of all methods for administering antiseptic remedies to the lung parenchyma. Intra-pulmonary injection is extremely limited in its scope. Intra-muscular injection is no doubt the best way to reach the blood and lymph, and there is no lack of suitable solutions to administer it this way, but it is not a pleasant mode of administration. Of methods by way of the alimentary tract, the mouth is evidently the most convenient gate, but its use has the serious objection of sometimes causing gastric disturbance. The rectum has met this drawback, and most drugs can be well absorbed by it, and if given in suppositories not much objection is made by the patient, hence it is a valuable alternative to the mouth in those cases where gastric disturbance arises. The phenols have been more used than any other antiseptics. Pharmacy has now reached a step beyond the crude drugs, carbolic acid, creosote, and guaiacol, which, from their caustic, poisonous reaction and unpleasant taste, were very difficult of administration, and has passed to compounds, which, while harmless and tasteless, are almost equally powerful as antiseptics; such are salol, betol, benzosol, paracresolol, guaiacol carbonate, creosote carbonate, and the group of bismuth phenols of which naphthol-, cresol-, and tribromo-phenol-bismuth are the most important. These phenols cannot be strictly termed antiseptics as regards living germs in the tissues, but there is good reason to believe that after absorption they combine with the albuminous toxines which result from germ life, forming non-toxic compounds which are eliminated in the urine as ethereal sulphates. They do not kill the bacilli, but

they obviate the evils resulting from the existence of these, and thus prevent injury to the tissues, the bacilli remaining harmlessly in the body, and in due course of time being excreted as effete material. There is no clinical proof of the extermination of the bacilli by the use of phenols, although there is ample evidence of the power they possess of keeping in check the evil symptoms of tuberculous septicæmia.—*The Birmingham Medical Review*, 1864, No. 189, p. 274.

The Treatment of Cancer of the Stomach.

Dr. H. Huchard cites among the anti-cancerous remedies, aristol, sodium chlorate and condurango. Aristol acts internally as does iodoform. Sodium chlorate diminishes vomiting, and hæmatemesis, increases the appetite and combats the cachexia; nevertheless it has no curative powers, and is contra-indicated in albuminuria. Condurango stimulates the appetite and the digestive power of the stomach when given as a wine or tincture or as a decoction. The diet must be regulated, hydrochloric acid administered up to the time of ulceration, then it must be replaced by pancreatin or pepsin. For the vomiting the choice of remedy must be made according to the emergency: morphine, cocaine, strychnine, lavage, gastric antiseptics, as sodium chlorate or salicylate, boric acid, naphthol, chloroform water, benzo-naphthol; nutritive enemata, as that of Leube, Flint, Dujardin-Beaumetz. The surgical treatment is not radical, for those operated upon succumb sooner or later to degenerative lesions of the mucous membrane inseparable from cancer; the patient dies; it is true that he dies cured.—*Journal des Praticiens*, 1894, No. 32, p. 379.

The Mechanical Treatment of Chronic Constipation.

Dr. F. Le Marinel believes that the mechanical treatment should be the treatment of choice in a large number of forms of chronic constipation. The manœuvres of massage comprise three forms—frictions, pressures, and percussions. In addition, various active and passive movements are undergone—flexion, extension, abduction, adduction, pronation, supination, rotation, and circumduction. The effects of medical gymnastics are studied as regards the circulation, respiration, animal heat, chemical phenomena, the muscles, bones and articulations, and the local results. The effect of massage upon the absorption, muscular system, nervous system, circulation, local temperature are fully recorded.

The technique of various authors is fully described and illustrated. The various forms of constipation which are likely to be benefited are—(1) from anæsthesia of mucous membrane; (2) from muscular paralysis; (3) from induration of the stools, and (4) from mechanical obstacle. The contra-indications are—(1) acute inflammation of intestines, peritoneum, peri-intestinal cellular tissue, or of intra-abdominal veins; (2) ulcerations (round, tuberculous) of stomach or intestines; (3) tumors of the alimentary canal (sarcomata, carcinomata, polypi); (4) voluminous fæcal masses of stony hardness. An interesting comparison is given of the value of diet, habit, purgatives, hydrotherapy, injections, electricity, various medicinal methods, with that described by the author, the general conclusion being that of all these

methods only one can be compared to the mechanical, and that one is electricity. But when one considers the question from the standpoint of success, massage shows figures which are not surpassed by electricity. So far as facility of application, massage is superior in that it does not require any apparatus, and can be applied equally well at the house of the patient as of the physician. Further, massage is better borne than electricity, and particularly by children. A series of 147 cases are reported, the treatment being that advocated in the paper. The care shown in the report, the apparent accuracy of diagnosis, the fulness of detail, give an especial value to the clinical histories. In the cases cited, those of the synoptical table, and eleven in detail, 158 in all, radical cure was obtained in about 90 per cent. The final conclusions are: 1. Mechanical treatment takes rank among those therapeutic agents whose action is most energetic upon the circulation, the respiration, and general nutrition. 2. It can modify the abdominal circulation, and cause certain foreign congestions, notably those which are met with in abdominal plethora, to disappear. 3. Under its action the muscles acquire an increase in volume and strength. 4. It is the best curative agent in constipation dependent upon muscular paresis or paralysis, if it is not of central nervous origin. 5. It is the best curative agent for constipation dependent upon diminished sensitiveness, or anæsthesia of the mucous membrane when it is due to local causes. 6. Finally, it is formally contra-indicated when constipation is due to acute inflammatory lesions or to tumors.—*Journal de Médecine, de Chirurgie, et de Pharmacologie*, 1894, fasc. 1 et 2, p. 1.

Poisoning by Chloralose.

M. Bardet has seen poisonous symptoms produced in a child of six years who had taken only three and a half grains of the drug. In two hours there were trembling, convulsions, later a cataleptiform condition, which lasted two hours. On awakening from this, all symptoms disappeared. This trembling and intellectual stupor has been observed in adults also, by Morel-Lavallée, after a dose of twelve grains. This remedy certainly ought to be used with caution.—*Journal des Practiciens*, 1894, No. 15, p. 178.

Intra-peritoneal Injection in Cholera.

Mr. Arthur Powell has used an apparatus which can, if necessary, be improvised from the armamentarium of the general practitioner. It is a forty-ounce bottle with a rubber stopper, through which are passed two pieces of glass tubing—one short, just reaching the neck of the bottle. To this a canula is attached by about four feet of rubber tubing, such as is used for infants' feeding bottles. The other glass tube reaches nearly to the circumference of the bottom of the bottle, its continuation outside being bent at a more or less acute angle. Its object is to allow the air to enter when the bottle is inverted and in use. It may, if desired, have a thistle funnel attached to facilitate recharging and to hold some sterilized cotton to filter the air. The fluid—a drachm of common salt to a pint of spring water—is boiled, filtered into the bottle and then re-boiled with the cork and glass tubes in position. Before inserting the canula into the vein the bottle is inverted until the

opening of the long glass tube is above the surface of the fluid, which will then run with a force proportionate to the height to which the bottle is raised above the canula. A hypodermatic needle can be attached for subcutaneous injection, or an aspirator needle can be used for injection into the peritoneal cavity. It is hoped that these operations may be more frequently performed now that it is possible to extemporize so simple and effective an apparatus.—*Indian Medical Gazette*, 1894, No. 5, p. 168.

Ergot or Hydrastis in Metrorrhagias.

Dr. Liégeois believes that ergot is the remedy for *post-partum* uterine inertia, but that hydrastis can be substituted with advantage in the treatment of metrorrhagias other than these. For chlorotics he prescribes ferric chloride or sulphate, but, with the iron, always hydrastis. For neurotic individuals the chloride or sulphate is replaced by the ferric valerianate or bromide and the hydrastis is continued, even during the menstrual epoch, because it does not give rise to uterine colic as does ergot. Neither ergot nor hydrastis can take the place of digitalis in cases where the hemorrhage is due to hyposystole in disease of the mitral valve of the heart; nor the place of quinine when it is due to malarial infection. Hydrastis is especially useful in the hemorrhages of the menopause; for the medication of urgency, hypodermatic injections of quinine are necessary. If uterine colic is a prominent symptom during the administration of hydrastis, the remedy is not at fault, but the cause lies in intra-uterine clots, and relief may be obtained with piscidia and viburnum. In metrorrhagias connected with uterine neuralgias and metritis he adds, to the hydrastis, piscidia, and viburnum, cannabis indica. For the hemorrhage of endometritis fungosa, or of cancer, surgical measures must be adopted. If the uterine hypercongestion is secondary to ovarian neuralgia the combination above mentioned is useful. He does not recommend hydrastinine, for the dose varies so greatly, nor is it advisable for hypodermatic injections, which are always painful and leave behind indurated nodes.—*Journal des Praticiens*, 1894, No. 29, p. 344.

[The combinations of drugs recommended in this paper have been in common use in America for nearly ten years.—R. W. W.]

Ichthyol Suppositories in the Treatment of Prostatitis.

Dr. A. Freudenberg has made use of this method of treatment in about thirty to forty cases since March, 1891. It is most useful in subacute and chronic cases, relieving the pain upon defecation, pressure, and other subjective symptoms, as well as the enlargement and hardening of the gland. The ammonium preparation was employed in dose from seven to fifteen grains in cacao butter, thirty to forty-five grains, as a rectal suppository, two being used each day. No observations upon prostatic hypertrophy are recorded.—*Centralblat für klinische Medicin*, 1893, No. 26.

The Action of Hydrochlorate of Scopolamine on the Eye.

Dr. Thomas R. Pooley gives his experience of the last six months. Scopolamine is obtained from the root of the *Scopolina atropoïdes*, and like atropine

and hyoscine, belongs to the pharmacological group of the tropeïnes, and as such the instillation of a water solution in the eye causes dilatation of the pupil. According to Landenberg, scopolamine, as well as hyoscine, are contained in hyoscyamus, without being identical with the latter; it is rather isomeric with cocaine, but yields different integral products. The conclusions are: That it is of value as a mydriatic and cycloplegic in the examination of anomalies of refraction; that its action is more complete than homatropine, and of about the same duration, and better than that of atropine sulphate, because its effects pass off sooner; that it is open to the objection that it produces toxic effects oftener than homatropine, in spite of statements to the contrary; that the temporary amblyopia sometimes induced does not seem to be of much moment; that in cases of short attacks of inflammation of the cornea, especially in some of the suppurative types, it is of special value. That this drug will soon replace atropine in the practice of ophthalmology is not well assured, but that it may prove a very valuable addition to the list of mydriatics which we now have, seems to be altogether likely.—*Therapeutic Gazette*, 1894, No. 3, p. 160.

An Antidote to Strychnine.

M. G. Grigorescu, in the course of some experiments to determine the action of toxic substances upon the excitability of peripheral nerves and muscles, developed the fact that butyl-chloral opposes the toxic action of strychnine. He found that if injections of strychnine were made (in frogs), those which received also the butyl-chloral remained torpid, while those with strychnine alone were tetanized; the least noise increased the tetanus of the latter, but the former did not show any spasm. After some hours the butyl-chloral was eliminated, and then these frogs were seized with tetanus, as were the others. On repeating the antidote up to the elimination of the strychnine, complete cures resulted. On experimenting with larger frogs similar results were obtained. The observations demonstrate that butyl-chloral energetically opposes its physiological action to the physiological action of strychnine.—*Archives de Physiologie*, 1894, No. 1, p. 32.

The Treatment of Skin Diseases by the Mineral-Oil Soaps.

Dr. J. Sümegh has made use of a soap which, on account of its containing naphtha, he has termed a mineral-oil soap. This soap possesses great advantages over the tar ointments, in that it does not act as an irritant. He has employed it for pityriasis versicolor, herpes tonsurans, mycotic eczema, ephelis, acne vulgaris, and commedones. It is used in seven different degrees of concentration in the following manner: The diseased area is moistened with lukewarm water and the skin rubbed with soap. The foam thus produced is allowed to remain five, and later, when the skin is not markedly reddened, ten, fifteen, or twenty minutes, then it is removed by washing, and the area dusted with powdered rice starch. If the skin is markedly irritated, the treatment should be omitted for a few days.—*Centralblatt für die gesammte Therapie*, 1894, Heft 5, S. 271.

[Composition and strength of the preparation are not given.—R. W. W.]

MEDICINE.

UNDER THE CHARGE OF

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THE CONDITION OF THE REFLEXES IN CASES OF TOTAL PARALYSIS OF MOTION AND SENSATION OF THE LEGS FROM AFFECTION OF THE SPINAL CORD.

DR. E. REYNOLDS (*Medical Chronicle*) states that it is only within the last few years that particular attention has been given to this subject, thanks chiefly to the labors of Dr. Charlton Bastian, Dr. Hughlings Jackson, Dr. Thorburn, Dr. Bowlby, and others, and now it is fairly well recognized and accepted as a clinical fact, whatever the interpretation may be, that in cases of complete transverse lesion of the spinal cord the knee-jerks are lost.

Reynolds' two cases are published to illustrate the fact that a spinal-cord lesion may cause complete paralysis and loss of sensation of the legs without total loss of the deep or even superficial reflexes.

The first is that of a laborer, aged thirty-nine, who was injured by a fall. Ten months after the accident there was total paralysis of motion and sensation below the level of the seventh intercostal space.

"*Reflexes.* Although tested many times, there is a total loss of the knee-jerk and the ankle clonus on both sides. No epigastric superficial reflex can be obtained, but the side abdominal reflex is present on both sides, but is feeble. The cremasteric reflex is normal and slightly more marked on the right side. On touching lightly or pricking the inner side of either thigh, there is an exaggerated contraction of the adductor longus of the same side. On tapping either ligamentum patellæ there is a marked contraction of the hamstring muscles, with flexion of the knee on the same side. Although this at first looked like a peculiar deep reflex I found that the same contraction could be produced even to a more marked extent by pricking the skin over the patella, showing that the reflex was superficial in character. On touching lightly, or better, by a very slight pricking of the leg below the knee, and especially the sole of the foot, there was at once a marked and exaggerated dorsiflexion of the same foot, with a flexion of the knee from contraction of the hamstring muscles. This was so marked that if the prick was more severe the contractions spread across to the other leg, and both feet were drawn up."

Case II. is that of a girl, aged thirteen years, with marked lateral curvature of old standing (? caries) in the dorsal region, causing total paralysis of sensation and motion below the lesion (from sixth interspace downward).

"*Reflexes.* These I have examined on very many occasions, and have always found as follows: Knee-jerks and ankle clonus extremely exaggerated; side abdominal reflex present; plantar reflex intensely exaggerated, so that the slightest touch or prick of a pin causes the leg (or even both legs) to be at once drawn up. The epigastric reflex is absent."

Two other cases of total loss of sensation and motion, with retention of some reflexes are alluded to. (Thorburn: *Medical Chronicle*, May, 1892, and Charlton Bastian, *Hysterical or Functional Paralysis*, p. 66). The writer does not suggest that these were cases of total transverse lesion of the cord.

Disinfection of Rooms.

For the disinfection of poor lodgings, in which tuberculous patients had died, Sheridan Delépine (Manchester) recommends solution of bleaching powder for the following reasons:

(1) The parts to be disinfected would necessarily be saturated with moisture; (2) chlorine, in the nascent state, would be generated where it was wanted, and much smaller quantities of disinfectant would be therefore sufficient; (3) there would be no necessity to use any complicated contrivance to secure the diffusion of chlorine, or to prevent its escape, though it might be well to keep the air saturated with moisture, to prevent the too rapid drying of the walls; (4) the assistants could apply the material without discomfort, and much less intelligence would be required on their part in the carrying out of their duties; (5) after the application of the solution, chlorine would continue to be evolved as long as all the chlorinated lime had not been decomposed, and that without anything further being required to be done after the first two or three hours; (6) the rooms would be fit for use as soon as dry again, and no poisonous substance would remain attached to their walls, as when perchloride of mercury is used; (7) if necessary, it was easy to increase its activity by adding acids to the solution, or by saturating the air of the rooms with acid fumes, and raising the temperature for a few hours."

Three series of experiments to demonstrate the efficacy of this method yielded entirely satisfactory results. The method of procedure recommended is as follows:

1. A solution of chlorinated lime (1 to 10) should be prepared. 2. The walls, ceilings, and floor should be washed with this solution, applied in the same way as lime or whitewash is usually applied. 3. This process should, for safety, be repeated three or four times in succession. By starting each time at the same corner of the room each layer would have time to penetrate into the paper and partly dry before the next is applied. 4. The room should then be closed as well as possible, a small, safe petroleum stove being first placed in the middle of the room, precautions being taken to prevent any chance of fire. Over this stove a large tin basin, full of water or chlorinated lime solution, should be placed. (By a simply devised water-bath arrangement a small capsule full of strong acetic acid or hydrochloric acid might be

placed over the boiling water, and in this way acidity of the air would be secured. This would cause a more rapid setting free of chlorine.)

"Chlorinated lime itself does not spoil things as much as one would expect, and can be used as indicated in rooms from which all hangings and carpets have been removed without any fear of damage, provided the walls and ceilings are not decorated with valuable paintings or papers. In the poor dwellings it is evident that this is not an obstacle to its use.[1] Small petroleum stoves, perfectly safe and giving much heat, can be obtained for a few shillings, and large tin dishes for a few pence."—*Medical Chronicle.*

The Starting-points of Tubercular Disease in Children.

In the *Lancet*, No. 3689, 1894, Walter Carr (London) communicates a paper on this subject, founded on the results of one hundred and twenty necropsies, and draws the following conclusions:

1. That tuberculous disease commences usually in the glands, the liability being at its maximum during infancy. 2. That tuberculous lesions in the cervical glands—as in the joints—might arise by infection through the blood stream, but that caseation of the bronchial and mesenteric glands, when primary, is usually, if not always, due to direct infection from the organ with which they were connected. 3. That tuberculous disease starts much more frequently in the thorax than in the abdomen. 4. That caseation of internal glands, from the frequency with which it is found after death, must have often existed alone and quite unsuspected. 5. That in regard to treatment, prophylaxis is by far the most important.

A Case of Pancreatic Colic.

Minnich (*Berliner klinische Wochenschrift*, 1894, No. 8, p. 187) has reported the case of a man, sixty-eight years old, without hereditary predisposition and with a good personal history, who at the age of forty became troubled with attacks of hepatic colic, which were attended with icterus and continued during a period of three months, but disappeared upon appropriate treatment. In the stools were found typical biliary calculi. There then followed a period of freedom from attacks for ten years and a half, when attacks of colic recurred; and these again yielded to suitable treatment. Some seventeen years later the man was suddenly awakened at night by an attack of colic resembling previous attacks. A second attack took place on the next day and a third several months later. At this time there appeared a sense of oppression in the epigastrium, together with loss of appetite and distaste for fluids. This condition persisted for about a month, without the occurrence of an actual attack of colic. At the end of this time the man was seized with diarrhœa, lasting for three days and ending with an attack of colicky pain referred to the left hypochondrium and to the epigastrium. The patient was well nourished and presented no œdema. The scleræ were a little yellowish and the skin dry and of normal warmth, but not icteric, although the seat of annoying itching and a chronic desquamative eczema upon the exterior sur-

[1] The quantity of bleaching powder required for a room measuring ten feet in all dimensions would not be more than six ounces, and the quantity of water three pints.

faces of the arms. The temporal arteries were slightly tortuous, but there was no other evidence of arterio-sclerosis. There was an absence of abdominal tenderness, so that deep palpation could be practised, but without yielding positive information. The liver was not enlarged, and the gall-bladder could not be appreciated. The spleen was likewise not palpable, and the area of splenic percussion-dulness was not increased. There was no evidence of dilatation of the stomach, and a peritoneal effusion could not be detected. The patient described the paroxysm as beginning with a sense of dull, heavy, constricting pain above the epigastrium and in the left hypochondrium, increasing in intensity and localizing itself deeply in at one point below the left costal margin just within the mammillary line. At the height of the attack the pain extended from this point circularly to the vertebral column in the course of the costal arch, thence radiating beneath the left scapula. At the termination of the attack pain was still perceptible at the point below the costal margin in an area about an inch and a half in diameter. Careful exploration of this region failed to disclose any morbid condition, excepting slight tenderness. The attack abruptly came to an end at the expiration of two hours, and was followed by a sense of hunger. The urine passed at this time was free from albumin and sugar, and did not respond to tests for biliary coloring-matter. From this time the attacks were repeated almost daily, usually setting in toward the end of the day and lasting from a fraction of an hour to several hours. The condition resisted the ordinary treatment for hepatic colic. An examination of the stools failed to disclose the presence of fat or fatty crystals or biliary calculi. On several occasions, however, light-gray, round calculi were found, which could be crushed between the fingers and microscopically proved to be constituted of amorphous matter. They dissolved in chloroform, leaving a turbid solution. They fused in the flame, giving off a dense vapor having an aromatic odor. The residue yielded the reactions of calcium carbonate and calcium phosphate. Subsequent attacks of pain occurred, but no more calculi or other abnormal matter was found in the intestinal evacuations. The condition was diagnosticated as one of calculous formation resulting in obstruction of the excretory ducts of the pancreas and giving rise to attacks of colic.

Recurring Symmetrical Local Asphyxia in a Young Child.

Israelsohn (*St. Petersburger medicin. Wochenschrift*, 1894, No. 16, p. 147) has reported the case of an anæmic and rhachitic child, three years old, that he was called to see on account of a deep-blue, almost black, discoloration of the fingers and toes of both hands and feet, of sudden onset. The discoloration extended from the tips of the fingers and toes to the wrist-joints and ankle-joints respectively, there terminating sharply. The affected extremities felt cold. The condition had existed for twenty minutes and had set in without appreciable cause, the child previously having been bright and cheerful. Consciousness was not affected, although the child appeared languid and apathetic. The heart-sounds were distinct, rhythmic, and pure. The radial pulse was 80 in the minute and small. The lungs presented no abnormality. A year before this attack the child had suffered with generalized œdema, without albuminuria, however, and this was attributed to the

anæmia. The condition disappeared in the course of a week upon a milk-diet, leaving anæmia and weakness. For the relief of the cyanosis frictions with spirit of camphor were directed, and in a short time the discoloration had disappeared. Two days later a precisely similar attack occurred, which again disappeared after friction with camphor-spirit. Four other attacks took place at intervals of two or three days, always soon subsiding under the influence of the frictions. It was observed, however, that the cyanosis in the lower extremities yielded when the friction was applied only to the upper. The case is believed to belong in the category of those described by Raynaud and known by his name, and in which the primary stage of local syncope escaped observation and the third stage of gangrene was averted. No importance is attached to the therapeutic measures employed, as it is believed that the paroxysms came to an end spontaneously after the spasm of the vessels from irritation of the bulbar vasomotor centres had relaxed.

The Effect of Compression of the Common Carotid Artery.

Hill and Moore (*British Medical Journal*, No. 1740, p. 962) found that on compressing the left common carotid artery there resulted at once sensations of formication and tingling, spreading down the right side of the body and accompanied with feelings of vertigo. A repetition of the experiment was followed by half a dozen clonic spasms of the right hand, which at the time lay upon the arm of a chair. The hand felt passively moved as if by some external agent, and consciousness of the spasms arose only from the peripheral sensations produced by the movement. The experiment was followed by vertigo and nausea lasting for some minutes. On compressing the left carotid against the spine at another time an undefinable sensation, as of numbness and prickling, arose in the left eye; this was followed by a distinct and rapid march of formication and numbness down the right arm, right leg, and then up the left leg. At this point the compression was withdrawn; the sensations, however, increased in intensity in the right hand, drew the attention to that part, and then the hand was felt to be passively twitched three or four times as if by some external agent. Nausea and vertigo followed and lasted for some minutes, and an acute feeling of dread of touching the region of the carotid again lasted for some days. In another experiment some difficulty was at first experienced in compressing the artery, and the first symptom produced was a succession of inspiratory spasms, which very probably were caused by stimulation of the vagus. Sensations in the eye of the same side followed and then three or four flexions of the left hand and fingers, which were at the time lying upon the knee. At the same time there was pallor and sweating of the face. The hand was felt to be passively moved by some external agent, and consciousness of the movement arose entirely from peripheral sensations. Vertigo and nausea followed. It is pointed out that the interest of these observations resides, first, in the realization of the intensely sensory side of consciousness. In all three of the experiments consciousness of the resulting movement arose entirely from the centripetal sensory impressions which had origin in the moving part. The central motor discharge was unaccompanied by any simultaneous consciousness of that discharge. Only the sensory part of the sensori-motor process

was accompanied by consciousness. Consciousness of peripheral sensations arose, first of all, as an illusion from the central stimulation due to the sudden anæmia; and secondly, from the real peripheral sensations due to the "passive movements" of the arm. In the second place, the experiments demonstrated the sensori-motor functions of the regions of the brain supplied by the middle cerebral artery. Finally, they fortified the teachings as to the origin of spasm in man from pathological changes in this region of the cortex.

Paralysis Agitans in a Young Person.

Lannois (*Lyon Médical*, 1894, No. 14, p. 465) has reported the case of a lad, eighteen years old, without neurotic predisposition, who from the age of twelve years, following an attack of measles, presented tremor, at first slight, but slowly progressive. The appearance of the lad was strikingly juvenile; his height and his general aspect, as well as his intelligence, were those of a child of twelve years. The face was round and fixed; both it and the pubes presented no indication of a growth of hair, while the genitalia were like those of a boy. There was no deformity of the head or curvature of the spine and no defect of the teeth. In walking the left leg appeared shorter than the right, but upon measurement no difference in length between the two was found. The tremor was regular and rhythmical and was most marked in the upper extremities, which were shaken in their entirety. The hands and the fingers, or the thumbs and fingers brought in apposition, were involved in rapid, tremulous movement, occurring from eight to nine times per second. The tremor was increased by emotion, by observation, and by exposure to heat or cold. It continued during repose, but ceased during sleep. It was lessened but not entirely subdued upon voluntary movement. The handwriting was exceedingly irregular. The tremor was evident in the lower extremities when the lad was recumbent. With the feet together the station was steady. If, with the patient in this position, the hands were placed upon the shoulders, a slight general shaking of the whole body could be felt, and this was transmitted to the head, but the head was not shaken independently; it was not moved when the patient was recumbent. There was no tremor of the chin, of the tongue, or of the ocular muscles. In walking or standing erect a characteristic attitude was assumed; the head was held fixed, the neck was slightly turned to one side, the trunk was inclined a little forward, and the arms hung by the side of the body, with the hands in supination and the thumb in apposition with the fingers. The movements in the fingers were intensified after the patient had been in this position for a short time. Complaint was made of a sense of heat and of undue perspiration, particularly at night. Speech was slow, but otherwise unaltered. Sensibility, in all its varieties, and the reflexes were intact.

Syringomyelia with Bulbar Symptoms.

Müller (*Deutsches Archiv für klinische Medicin*, B. lii., H. 3, 4, p. 259) has reported the case of a metal-moulder, eighteen years old, without neuropathic predisposition, who, three years before coming under observation, was seized with vomiting, in the absence of assignable cause. The vomiting continued for three or four weeks, often being repeated several times daily, but finally

ceasing spontaneously. Some four months later some difficulty was noted in grasping and holding objects with the right hand, and this was the more marked when the hand was not in sight; there was also some defect in recognizing objects by touch. Some time after this a sort of numbness appeared in the right lower extremity. After the lapse of eight months headache would frequently occur, of variable intensity and duration. Then vertigo became manifest, particularly on looking upward and when lying recumbent. Difficulty in deglutition set in a short time later. Solids were swallowed with difficulty; liquids returned through the nostrils. There was at no time fever. Dyspnœa occasioned some distress. The patient continued at work for thirteen months after the appearance of the first symptoms; then he was compelled to stop on account of the vertigo, difficulty of gait, and diplopia. At this time it was said the face was distorted, the right eye turning inward. There was a sense of loss of control of the right lower extremity, which felt as if it were attached to the trunk by artificial connections. Some improvement now followed, inasmuch as the patient was able to walk better and for a longer time. When held dependent there was some formication in the fingers of the right hand and in the arm. Shooting pains were felt upon the whole right side of the body, compared by the patient to electric shocks. There was evidence that the appreciation of position was lost in the right hand, for when an attempt was made to reach this member with the left hand the former was not found in the position in which it had been thought to be. For a month there had been a disagreeable sensation referred to the left upper extremity, most nearly comparable to a sense of heat. This extended up over the neck to the ear. For a month, too, there had been some difficulty in flexing the fingers of the left hand. The little finger was additionally abducted, and from time to time was involved in involuntary movements of flexion. Wasting and hardness of the ball of the left thumb had been observed. There was no history of alcoholism, infection, or abuse of tobacco, and none of traumatism or of undue exposure to cold. The only cause suggested by the patient for his condition was excessive muscular effort in his work. He had repeatedly burned himself without pain and without consciousness of the occurrence except from the presence of blisters. There was nasal deviation of the right eye, from paralysis of the sixth nerve. The pupils were equal and reacted to the usual stimuli. The fundus presented no lesion. The nasal half of the visual field of the right eye was defective, from the internal strabismus. The muscles supplied by the right facial nerve were paralyzed; the electric reactions were unaltered. In the distribution of the trigemini, both the temperature-sense and the pain-sense were impaired, though common sensibility was preserved. The right half of the velum pendulum palati was paretic. Speech was nasal. The right recurrent laryngeal nerve was paralyzed, as indicated by the cadaveric position of the corresponding vocal band. The reflex irritability of the epiglottis, the ary-epiglottic folds, and the interior of the larynx was much diminished, although sensibility was preserved. The right half of the tongue was smaller and less active than the left, and was the seat of fibrillary tremor; its electro-contractility was diminished; when the organ was protruded it deviated to the right. Reflex irritability of the uvula, the root of the tongue, and the posterior wall of the pharynx was lost. The spinal column was deviated to the right in the dorsal region.

There was a sense of itching in the interscapular region. The thenar and hypothenar eminences, and the interosseous spaces were wasted. Gross power seemed to be alike in both hands. Fine movements were not performed with the right hand so well as with the left, and less well when the eyes were closed. A similar defect of co-ordination was present in the right lower extremity. Jerky, irregular twitching movements occurred in the right upper extremity when hanging dependent and not kept in sight. A similar condition existed in the right lower extremities. From time to time rapid movements of flexion would take place in the little finger of the left hand. There would also occur attacks of pain radiating from the right side of the neck to the fingers of the right hand. The entire left upper extremity was the seat of a peculiar, uncomfortable feeling extending over the left side of the neck to the ear, and compared by the patient to a sense of heat. All forms of sensibility were impaired, and there was analgesia in an area occupying the right side of the neck, the right shoulder and arm, and the upper part of the chest on the right side, while in a corresponding area upon the left side the temperature-sense and the pain-sense were lost. Below these areas to the level of the nipples the temperature-sense only was lost. There was analgesia, together with inability to distinguish sharp and dull points in the right hand and fingers, and the sense of localization was also defective. The appreciation of both active and passive movements, as well as the sense of position and the sense of weight, was defective upon the whole of the right side of the body. There was an inability to recognize objects with the right hand by means of touch alone, without the aid of the vision. The limits of the areas of thermo-anæsthesia and analgesia were bounded by zones of thermo-hypæsthesia and hypalgesia respectively. Electro-sensibility was impaired in the right upper extremity as compared with the left. The gait was ataxic, the station unsteady. A subcutaneous panaris that formed on the little finger of the left hand was incised without causing pain. The reflexes in the lower extremities were exaggerated. While under observation some of the symptoms remained stationary, while others underwent varying degrees of improvement.

Infectious Purpura.

At a recent meeting of the Société Médicale des Hôpitaux, Lebreton (*La Médecine Moderne*, 1894, No. 8, p. 126) reported the case of a girl, eighteen years old, who, forty-eight hours after violent emotion, presented grave general symptoms: intense headache and backache, chills, fever, and repeated epistaxis. Twelve hours later there appeared upon the hands, the arms, the thighs, purpuric spots of confluent arrangement, and so numerous as to leave scarcely an area of clear skin. Upon the body, however, the eruption was discrete. During a period of forty-eight hours the condition appeared alarming, and ether and caffein were injected subcutaneously and oxygen administered by inhalation. The spleen was enlarged and the periphery of the body was cold. In the course of several days the threatening symptoms gradually subsided, and recovery finally took place. Cultures prepared from the blood and from the urine with suitable precautions disclosed the presence of the staphylococcus pyogenes albus. The portal of entry of the micro-

organism was, however, not evident. Neither the patient nor those about her presented any suppuration, or furunculosis, or impetigo, nor she herself an angina. In the discussion WIDAL pointed out that, as in other cases of infectious purpura streptococci and pneumonia-cocci had been found as well as staphylococci, the question arose if the presence of these might not be secondary to some primary infection. MATHIEU called attention to the possibility of mistaking for hemorrhagic variola such a condition, attended with a copious purpuric eruption and profound constitutional symptoms. Furthermore, in the absence of a bacteriologic examination, the case, following emotional disturbance, would have been considered one of neuropathic origin.

THE RESULTS OF PROGRESSIVE TRANSPLANTATION OF THE THYROID GLAND IN THE DOG.

GODART (*Journal de Méd., de Chir., et de Pharmacol.*, 1894, No. 4, p. 49), as the result of a rather extended series of experimental observations, concludes that the symptoms observed after removal of the thyroid gland are not to be explained upon the supposition that the normal gland exercises a regulatory action upon the circulation. Nor is there support for the belief that the abnormal manifestations are due to a lesion of the nerves of the gland or adjacent to it. An adequate explanation of the symptoms is, however, to be found in the loss of the cellular activity of the gland, as demonstrated by the results of grafting or transplantation. The unpleasant results that follow thyroidectomy are to be avoided by inserting grafts before the removal of the gland, and by making this removal gradual and not abrupt.

SURGERY.

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THE CURATIVE EFFECT OF EXPLORATORY LAPAROTOMY.

SHEPHERD has added another to the now rather extended list of cases in which simple exploratory incision has been followed by marked amelioration or cure of apparently grave conditions (*Montreal Medical Journal*, 1894, No. 9). Many such cases have been recorded by reliable authors. The explanation of the beneficial results following exploratory operations must for the present remain problematical, and has been carefully avoided by most authors. In

1891, White collected a large number of these cases (*Annals of Surgery*). In this paper he suggested that the effect of the anæsthetic, psychical influence, relief of tension and reflex action, might be possible factors. The cases are too numerous to be attributed to accident or coincidence.

The author's case was that of a woman, aged twenty-eight years, with a tumor in the neighborhood of the umbilicus. Her morning temperature was 100°, and the evening temperature 101°. Every second day she had a severe sweat. The growth was about the size of an orange, smooth, tender, and movable with the respiration. The nature of the tumor was difficult to determine, as there was nothing in the history bearing on the affection. At the operation the tumor was found to be connected by a broad base. It was decided not to remove the growth. The wound was closed and the patient made an uneventful recovery. After the exploratory incision she had no more tenderness, and after the first day or two no more pain. The sweating ceased and the temperature became normal. The tumor disappeared within a year after the operation, and the patient continued in perfect health.

Paralysis of the Serratus Magnus and Dislocation of the Scapula.

Moorhouse (*British Medical Journal*, 1894, No. 1726) records the following rare case: A miner complained of pain in the region of the deltoid and right side of the chest. The symptoms disappeared by rest. The pain returned again, however, and the shoulder-blade was growing prominent. Examination revealed two painful spots—one behind the deltoid muscle, corresponding to the position of the circumflex nerve, and the other to the inner side of the biceps. The pain was only marked when he held the forearm out between pronation and supination. With the arms hanging at the side, the lower angle of the right scapula was half an inch nearer the spine than the left. With the arms stretched out in front, the right scapula came out like a wing, to such an extent that the dorsal border was two inches from the chest wall. The distance between it and the spine was only two and one-fourth inches as compared with five on the left side. The left arm could be stretched out an inch and a half further than the right, and the affected side was very weak. A curious feature was that when the place of the serratus magnus was supplied by pushing the scapula forward against the chest the pain ceased. The right shoulder was lower than the left and the deltoid considerably atrophied. The right half of the chest moved much less than the left on forced inspiration. It would seem that there was some affection of the circumflex and posterior thoracic nerves, the paralysis of the posterior thoracic nerves having been followed by the characteristic dislocation of the scapula. No cause could be given. Under the influence of a tight belt, fly-blisters, stimulating applications, and hypodermatic injections of strychnine into the muscle, recovery was complete at the end of two months.

The Treatment of Peritonitis by Drainage.

Barling records (*British Medical Journal*, 1894, No. 1725) the histories of ten cases of peritonitis treated by drainage. These include cases of acute sero-purulent peritonitis, purulent peritonitis, and tuberculous peritonitis.

Although four of the patients died, the author considers the results satisfactory.

While the diagnosis of peritonitis is usually easy, cases are occasionally met with which are very misleading. Instead of the constipation generally present, there may be diarrhœa; instead of a hard wiry pulse, it may be soft and dicrotic; distention and tenderness may be absent. The most reliable signs in the author's experience are the steadily increasing frequency of the pulse, often with a low or falling temperature, and the facial expression, this being either pinched and anxious or flushed, the latter usually being associated with delirium. In many cases the phenomena are those of septicæmia, which mask and obscure the symptoms immediately due to the inflammatory process in the peritoneum. These latter are pain, shock, paresis of the bowel, and a hard pulse, and are mainly produced through the impression made on the widespread sympathetic plexuses of the abdomen by the inflammation which envelops them. It is worth noticing that the umbilicus seems to be the point at which inflammatory collections naturally find their way out, if they are general.

Aseptic and Septic Surgical Cases with Special Reference to the Disinfection of Skin, Sponges, and Towels.

Lockwood has added another contribution to this subject (*British Medical Journal*, 1894, No. 1726). This paper is a continuation of two previous reports which appeared in the issues of the same journal of October 25, 1890, and May 28, 1892. The investigations have been conducted on the same line as laid down in those communications. The difficulty of rendering the skin sterile, as has been so often remarked upon, is emphasized by the present investigations. In a number of operations the skin was disinfected with the greatest care, after the most approved modern methods, including vigorous scrubbing with soap and warm water, followed by carbolic lotion 1:20, bichloride lotion 1:1000 and 1:500, solution of potassium permanganate and solution of oxalic acid; notwithstanding, small portions of skin dropped into culture-tubes invariably resulted in the growth of one or more of the various bacteria which infect the skin. Some of the operation wounds, however, ran a sterile course. The comparative inefficiency of corrosive sublimate and carbolic acid to inhibit bacterial growth was demonstrated by mixing solutions of these chemicals, in different proportions, with broth cultures, and it was found that inoculated colonies continued to grow in these media. The failure of watery solutions to penetrate the pores of the skin has been recognized for some time, and an effort made to overcome this defect in the technique by employing alcohol, ether, or benzine. In view of this fact the author was induced to employ glycerin as the vehicle for applying the chemical antiseptic. Accordingly bichloride in glycerin 1:2000, and carbolic acid in glycerin 1:40, were applied after the usual preparation of the skin, twenty-four hours before operation. This plan was more successful. In about half of the cases test-tubes inoculated with fragments of skin so prepared remained sterile. Inasmuch as biniodide of mercury is not precipitated by albumin, it was thought that this substance might yield more satisfactory results. Of two trials, however, the skin was found by culture-

experiments to have been aseptic in one and to contain staphylococcus albus in the other.

Of 21 experiments upon the disinfection of the skin which have been carefully detailed, 7 were successful—in 6 the wounds healed by first intention, without either local or constitutional reaction. In 14 of the cases the skin was septic—9 of these healed by first intention and 5 suppurated. In none of these, however, was the suppuration severe or prolonged.

In regard to disinfection of the hands, the author states, the best results have followed the usual thorough scrubbing with soap, hot water, and nail-brush, cleansing the nails and then immersing for one minute in a 1 : 1000 solution of sublimate in alcohol.

The observation on the disinfection of towels is interesting: Three of four towels which had been soaked for two hours in 1 : 20 carbolic solution were found to contain staphylococcus pyogenes albus. In another case a towel which had been kept in carbolic solution 1 : 25 for twenty hours grew a bacillus. In another case a towel which had been immersed for twenty-four hours in a solution of the same material was sterile. The result of the use of bichloride was also not uniform. Of 6 experiments in sterilizing towels by the steam sterilizer for a half-hour, 5 were successful and 1 failed. The latter was attributed to the towel having been tightly folded during the sterilizing process. By taking the precaution of placing the towel loosely in the sterilizer, the author believes this method may be relied upon; the towels being kept, of course, in antiseptic solutions after being taken from the sterilizer.

The sponges were prepared as follows: If new they were well shaken, then soaked in a solution of hydrochloric acid (1 drachm to a pint) for twenty-four hours. Next they were thoroughly washed and squeezed out in water at the temperature of 100° F.; and then immersed in a cold solution of sulphurous acid (1 : 5) for twelve hours, being kept below the surface of the water. Lastly they are squeezed as dry as possible and placed in carbolic solution 1 : 20, ready for operation. Of 12 experiments with sponges so treated, 11 were found to be sterile, the failure having occurred when the process was carried out by an inexperienced person. As carbolic acid evaporates rapidly, the jars containing the sponges must be tightly stoppered. These experiments show that asepsis is with the greatest difficulty obtained by the use of chemicals, but once secured, either by this means or by heat, dilute solutions of chemicals suffice for its continuance.

Operative Intervention in Anuria of Calculous Origin.

Demons and Pousson have collected (*Annales des Maladies des Organes Génito urinaires*, 1894, No. 2) from literature, fifteen cases of calculous anuria which were subjected to operation, to which they add three cases personally observed. Of the eighteen cases, six died—a mortality of 33⅓ per cent. This compares very favorably with those treated expectantly. Legueu places the mortality at 71.5 per cent. in cases treated without operation.

The authors conclude that calculous anuria indicates operation as urgently as does intestinal obstruction. The calculus must not be attacked, however, from in front. The uncertainty regarding the exact location of the obstruc-

tion contra-indicates this course. The creation of a means of escape for the urine above the obstruction greatly enlarges the field of action. Access to the renal pelvis is to be obtained by an incision through the convexity of the kidney. This operation, being so simple and free from danger, is to be preferred to ureterotomy or pyelotomy, unless the location of the calculus has been accurately determined. Calculi are usually arrested at the mouth of the ureter, or at its upper extremity. In this way nephrotomy often becomes curative, as it permits of the extraction of the offending calculi, either directly or by pushing from below upward or from above downward, or by retrograde catheterism of the ureter. Of six cases of calculous anuria treated by nephrotomy, two terminated fatally on account of a too tardy intervention. The four remaining cases were cured. The urine began to escape by the lumbar opening immediately after operation, and resumed the natural course in from one to thirty-five days, the calculus having fallen into the bladder. The large incision into the kidney for calculous anuria is justified by the pathological physiology.

The Effect of Iodoform on the Tissues.

L. v. Stubenrauch has recently completed an exhaustive experimental study on this subject (*Deutsche Zeitschrift für Chirurgie*, 1893, B. xxxvii., Hefte 5 u. 6). The investigation was suggested by the very frequent use of iodoform in local tuberculous affections and the good results so frequently observed. Many writers hold that the effects noted in these cases are due to the anti-bacillary action of the drug, by which the tubercle bacilli are destroyed. The effect of the iodoform on the tissues had not been studied, however, and Stubenrauch therefore determined upon the work of which this communication is the outcome.

The effect of iodoform upon the different tissues of the body, both normal and pathological, were carefully investigated. Observations upon man have been compared with similar experiments on animals.

The work is divided into four parts: 1. Historical; 2. Bacteriological; 3. Chemical; and 4. Histological.

The author arrives at the following conclusions:

1. Iodoform is not an antiseptic in the true sense of the term, as is corrosive sublimate, inasmuch as it does not destroy bacteria by short contact. The investigations heretofore made upon several different bacteria have shown that iodoform only inhibited the growth of milzbrand and tubercle bacilli.

Iodoform probably possesses no specific anti-tuberculous effect. Experiments on pure cultures of tubercle bacilli have repeatedly shown that when the iodoform remained in contact for a long time the growth was arrested, but it was not possible to destroy the vitality or growth of fully virulent tubercle bacilli by contact with iodoform in the animal body.

The favorable results which follow the iodoform treatment in cases of chronic tuberculous abscesses must not be taken as unconditional proof of the specific anti-tuberculous (anti-bacterial) effect of iodoform. The bacilli which are contained in tuberculous abscesses die under the influence of iodoform, but it is not proven that the destructive effect is primarily due

to the iodoform; there is a greater probability that the effect of the latter on the tissues, in connection with the experimentally proved slight anti-bacterial effect, has caused the destruction of the bacilli. Iodoform interferes with bacterial growth—if ever—in the undecomposed as well as in the decomposed condition.

2. The decomposition of iodoform can take place outside of the body as well as inside.

a. In solutions (ether, alcohol, or oil) or mixtures (water, gum, or glycerin) which are exposed to the sunlight. In the mixtures, decomposition of iodoform takes place in a warm place even if light be excluded, the rapidity depending upon the degree of the temperature. The effect of live steam on iodoform mixtures or emulsions is to separate certain quantities of iodine (and hydriodate) and form new compounds, or they remain free, according to the nature of the different organic substances which are present. The quantity of iodine which becomes liberated from iodoform at 98° or 100° is according to the nature of the several vehicles, and the greater or less excess of air, but is independent of the form of the preparation. Considerable decomposition takes place in iodoform by the heat of live steam in glycerin.

b. Iodoform is further decomposed outside of the body by the liberation of hydrogen, and further through reducing substances—as, for example, powdered zinc, silver—and also through neutral nitrate of soda (with the addition of heat), and finally through the products of the bacteria (ptomaïnes). Inside of the body there is decomposition of iodoform in considerable degree, especially in wounds in which there is active reduction (and where ptomaïnes are also present), in the cellular tissue under the skin, in the intestinal tract, the great cavities of the body, etc. With the albumin of the tissue juices the iodoform unites to form iodalbumin, which when liquid may be absorbed; if not, it decomposes further. The iodoform leaves the body in the form of soluble combination. The local as well as the general effect of the iodoform is a protracted iodine effect.

3. Iodoform acts on the tissues of the body, however, only if it is decomposed. Locally as well as from the blood it acts on glandular organs to such a degree that degenerative decay of the epithelial elements takes place. In this way the local application of iodoform to the kidney produces a degeneration (fatty) in the epithelium, and also a decay (fatty) of the cells in the kidney, liver, and other organs, if the iodoform has produced a general effect. All pathological new formations are influenced in a high degree by iodoform. Iodoform stops the formation of giant cells in the tissues in the neighborhood of a foreign body.

In serous cavities it causes coagulation of albumin, which, if the conditions of absorption are favorable, soon disappears. If the iodoform becomes decomposed and if certain products of decomposition remain for a long time in the cavity in consequence of unfavorable conditions of absorption, as in hydrocele, etc., then these (products of decomposition) are capable of causing an inflammation in the walls of the cavity. The effect of this is a transudation which coagulates, becomes organized, and cure results by the formation of connective tissue.

On tuberculous tissue iodoform acts probably by hastening the decay of the epithelioid cell collections which are destined to degeneration, while the more

resistant of the epithelial elements is prepared for a change into healthy tissue.

Echinococcus in the Sac of a Hernia.

Koslowski reported in the *Chir. Annalen*, 1893, p. 609 (*Centralblatt für Chirurgie*, 1894, No. 10), the following case: A man, thirty-four years of age, a blacksmith, applied for advice on account of a tumor in the groin which appeared on the previous day. The man had previously complained of undefined pains in the abdomen. On examination, a tumor as large as a small hen's egg was seen below Poupart's ligament. It was smooth, hard, flat on percussion, and its origin could be traced back into the inguinal canal. The patient said the swelling had frequently appeared before. This he reduced himself by pouring a little alcohol in a saucer, lighting it, and lying down by the flame, so that the tumor became warm. On this occasion this procedure failed. Strangulated inguinal hernia was suspected, and taxis attempted under ether. This failing, operation was performed the following morning. On cutting down, a vascular layer of fat presented which much resembled omentum. To clear up the matter, the sac was dissected free, with some trouble, and incised. The incision into the wall of the sac revealed a cyst as large as a pigeon's egg, a smaller cyst adjoining. The cyst proved to be echinococcus. A small mass of omentum was found and reduced. The neck of the sac was ligated and removed. The patient left the hospital on the twenty-sixth day, well. The author supposes that the echinococcus formed in the omentum.

OTOLOGY.

UNDER THE CHARGE OF

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Three Cases of Unintentional Opening of the Lateral Sinus.

Dr. J. E. Sheppard, of Brooklyn, N. Y., in the *Archives of Otology*, vol. xxii. No. 3, gives an account of three cases of unintentional opening of the lateral sinus. In the first case there seems to have been entire absence of a bony wall between the mastoid and cranial cavities. By the perforation of a membranous diaphragm between these cavities a hemorrhage occurred. In the second case the flow of blood came after removing a pledget of cotton used for drying-out the cavity. It seems probable that with the curette a piece of necrotic bone was made to wound the sinus wall, but was not sufficiently displaced to allow of hemorrhage until the cotton was removed. There were no disturbing symptoms, and the wound healed entirely in a comparatively short time. In the third case, the author says, it is difficult to explain how the sinus was opened. He suggests, as an explanation, that the curette may have splintered the inner plate, the posterior end of the splinter penetrating the sinus wall.

Trepanation of the Skull and Mastoid Process in Consequence of Suppurative Otitis.

Polo (*Révue de Laryngologie et d'Otologie*, vol. xiii., No. 1) records the case of a child, six years old, the subject of suppurative otitis media following measles. There was a rapid development of headache, coma, dilated pupils, vomiting, left internal strabismus, contraction of the muscular fibres of the face and of the orbicularis muscle. There was also a perforation of the membrana tympani. The mastoid process was painful, but its aspect was normal, and there was no paralysis of the limbs. Polo first performed trepanation of the mastoid process without any result. On the next day he trephined the skull in the temporo-sphenoidal region, puncturing the brain forward and upward without any success. Pus was finally discovered in the centre of the temporo-sphenoidal lobe at the depth of 4 cm., and the abscess was opened by a bistoury. The child died the following night in coma. After the operation the left eye, which had been turned inward, deviated outwardly.

Extensive Necrosis of the Temporal Bone of a Child; Mastoid Operation; Death from Exhaustion and Hemorrhage.

Dr. Knapp, of New York, read a paper under the above-named title at the meeting of the American Otological Society (*Transactions*, vol. v.) The patient was a feeble, anæmic child. For a few months there had been an offensive and copious otorrhœa of the left side. The mastoid region was swollen and soft, but no fluctuation was present. The mastoid was exposed, but there was no subperiosteal collection of pus. The outer table of the mastoid was then removed, exposing a large cavity filled with necrosed bone and granulations bathed in pus. This was removed and the cavities packed with corrosive-sublimate gauze. The child improved to some extent, but there was no tendency to repair. The child sank and died in the course of a few weeks.

Notes on Sixty Cases of Disease of the Mastoid Process in which the Antrum was Opened.

Dr. Adolph Bronner, of the Bradford Infirmary, England, during the last year has operated upon a number of mastoid cases. Of the 60 cases referred to, 19 were under ten years of age, 20 under twenty years, 12 under thirty years, and 8 over thirty years of age; 39 were males, 21 were females; 25 were acute and of under one month's duration. In 17 cases there was an external fistula. In 21 there was a mastoid abscess. In 10 the mastoid process was thickened. In 8 there was no swelling, but pain on pressure, and in 4 there were no objective or subjective symptoms whatever in connection with the mastoid process.

In 45 cases there was either a fistula or some superficial caries of the bone found at the time of the operation, and in 15 cases chiselling to some depth was necessary before striking pus or the antrum. In 8 cases the operation failed to relieve the symptoms and the patient died in from one to fifteen days. The ear disease was due to influenza in no fewer than 5 of these cases.

Brain abscess was the cause of death in 2 of these cases, meningitis in 3, and disease of the lungs in 3, in 2 of which were found thrombus of the lateral sinus, and in 1 the sinus was normal.

Of the 52 cases which were relieved, 3 showed signs of disease of the lungs, in 2 of which the mischief was diagnosticated before the operation; 36 of the 52 were cured within one year, that is, the discharge from the ear had ceased, whereas 9 still had a discharge at the end of one year, and 7 were lost sight of. That the discharge still continues in a very large number of cases is, in the opinion of Dr. Bronner, due to the fact that the after-treatment is so frequently neglected.—*British Medical Journal*, vol. ii., 1893.

Symptoms and Treatment of the Septic Infection of the Lateral Sinus, as Illustrated by Ten Cases.

Mr. W. Arbuthnot Lane, in the Section of Otology of the British Medical Association, presented a paper based on ten cases of septic infection of the lateral sinus on which he had operated. In every case the suppurative disease of the ear was the origin of the infection of the sinus. Fourteen very interesting conclusions follow, which want of space obliges us to omit.—*British Medical Journal*, vol. ii., 1893.

The Treatment of Otitic Brain Abscess.

Kretschmann (*Münchener medizinische Wochenschrift*, 40. Jahrgang, No. 29) gives an account of two cases of otitic brain abscess. The first was of special interest because of its occurrence in consequence of an acute suppuration of the middle ear. On account of the absence of characteristic symptoms the abscess was not discovered until the autopsy.

The other abscess of the brain developed in connection with a chronic suppuration of the middle ear and was cured by operation.

Discussion on the Surgical Treatment of Mastoid Disease and its Complications.

The above-named discussion was conducted in the Section of Otology of the British Medical Association at its meeting in Newcastle-on-Tyne. The discussion was opened by Prof. Macewen, of Glasgow, who said that the title of the subject for discussion meant the extension of middle-ear disease to the mastoid region, and that it included for the most part infective disease of the middle ear, and excluded tubercle and carcinoma. He further said that he had found that the greater majority of these diseases travelled from the middle ear to the mastoid antrum and cells, after that invading the mucous membrane, the bone, the dura mater, and membranes of the brain. In many instances, after erosion of the bone, masses of granulation tissue extruded themselves upon the dura mater, which, on removal, promoted re-infection by a fresh surface coming in contact with the infective material pent up in the middle ear. In infective purulent disease of the middle ear the mastoid, antrum, and the mastoid cells ought to be thoroughly ablated. As a rule, after opening the antrum the attic of the middle ear was exposed by enlarging and opening the osseous parts in front. After exposing the

attic, the ossicles are to be removed, and the whole of the granulation tissue of the middle ear cleared out. After this the tegmen of the attic should be scrutinized by efficient light, and if eroded fully opened up, the granulation tissue removed from the dura mater and the brain laid bare, and if necessary opened into. He then spoke of further treatment of brain abscess and of disease of the sigmoid sinus and infective thrombus of this sinus.

Mr. Victor Horsley said that his experience of such cases coincided with almost every point Prof. Macewen had advanced. He said the most important question among those that he would consider, at least as far as the interests of the patient were immediately concerned when the case was one of simple otitis media purulenta, was the question as to how long effort was to be made by ordinary antiseptic treatment to get the cavity to heal up before the surgical operation of clearing the tympanum and mastoid was undertaken. He suggested that one year would be the limit, and that if the discharge had not subsided, and if cicatrization did not occur within that period, the antrum of the mastoid should be laid open and the opening continued into the tympanum so as to make one space of both cavities. In this way, he suggested, the grave risks to life of persistent discharge from the ear, such as blood-poisoning, destruction of the bone, thrombus of the sinuses, inflammation of the membranes of the brain, and cerebral abscess could be avoided.

Mr. Hugh E. Jones, of Liverpool, then considered a method of determining the condition of the mastoid before operating. He said that in the vast majority of cases the tuning-fork was heard best when placed over the small depression, called by Prof. Macewen the supra-meatal triangle, which existed just below the posterior zygomatic ridge and a little above and behind the bony osseous meatus. In a few cases the sound appeared to be nearly equal over the whole mastoid, but at the upper and posterior margins well defined. In one such case, in which the sound was unusually well conducted, on opening the mastoid it was found to consist of one large cavity which was filled by cholesteatomatous masses. The same auscultatory result was obtained in another case of cholesteatoma of the meatus and tympanum. In cases in which the sound was diffused, but not unusually loud, or perhaps less loud than usual, he had found a diploëtic or sclerosed mastoid with a small deep-seated antrum.

Dr. William Hill in his remarks said that in most of the specimens which he presented, the postero-supra-meatal triangle with its pit could be well seen, and when this portion of the bone was removed, as in some of the bones shown, the antrum was found at a variable depth beneath. He thought the term, mastoid antrum, a misnomer, because the antrum was bounded above and externally by the squamosal bone, anteriorly by the petrous bone and inferiorly only by the mastoid portion of the petro-mastoid bone. The term temporal antrum appeared to be free from objection.

Dr. William Robertson gave as his opinion that chronic discharges from the ear favored infection, which might lead to invasion of important structures lying close at hand. He agreed with Prof. Horsley that one year should be the limit of antiseptic treatment for the cure of chronic otitis media purulenta.

Mr. Rushton Parker, of Liverpool, advocated the routine use of car-

bolized glycerin, one in twenty, as suggested to him by Mr. Lowe, of Bath, in preference to antiseptic injections which could not act thoroughly before the mastoid opening was made and tended to drive inward, without removing, septic agents and products confined in the bony cavity.

Dr. William Milligan, of Manchester, advocated opening the tympanic attic, and its exploration in cases of chronic suppurative middle-ear disease at the same time that the antrum was opened. The attic offers such an admirable space for the production and propagation of septic material that it should be the surgeon's aim to clear it thoroughly of all its putrid contents.—*British Medical Journal*, vol. ii., 1893.

[So true is this that it is highly probable that many cases of antro-mastoid suppuration could be prevented by an early removal of diseased ossicles in chronic suppuration of the middle ear, especially of the attic.—Rep.]

The Treatment of Encephalic Abscesses Consecutive to Suppuration of the Ear.

A. Broca (*Gazette hebdomadaire de Médecine et de Chirurgie*, Année xl., No. 38) under this title gives a general review of this subject from the time of Lebert down to the present time. After considering at some length the opinions of Barker, Stoker, Bergmann, Hare, J. Lloyd, Macewen, MacBride and Miller, Chauvel, Gull and Sutton, Krishaber, Pitt, and others, he says: "Being given a chronic suppurative otitis media, accompanied with cerebral symptoms not well determinable if there is a mastoid fistula or symptoms of mastoiditis, it is necessary to perform generous trepanation of the mastoid and of the tympanic cavity. The same thing should be done even when the mastoid region appears normal externally, for sometimes an unsuspected retention of pus will be found in it which causes the symptoms, and also because, if it is necessary to go further, the mastoid path is the best way of proceeding.

Osteoplastic Operations on the Skull, with Report of a Case of Cerebral Abscess following Otitis Media cured by Operation.

Dr. Brentano, in *Allgemeine medicinische Central-Zeitung*, No. 26, gives an account of a brain-abscess in a patient who had suffered from otorrhœa since childhood. Lately, following a cessation of the discharge, headache, constriction of the head, stiff neck, irregular pulse, somnolence, and tenderness of the occiput manifested themselves. The mastoid process was opened, and its cells found to be in a state of suppuration. After removal of the pus the dura was exposed and found to be black in color at the upper part of the cavity. As only temporary improvement occurred, another endeavor was made to find the suspected abscess beneath the discolored dura mater. At this point an abscess was discovered and emptied. Undisturbed recovery then took place.

Trepanation for Abscess of the Brain.

Terrillon communicates the history of a case of trepanation undertaken or relief of an intra-cranial abscess consecutive to chronic suppuration of the ear. The operation was performed eighteen months before it was reported,

and the cure appears to have been complete. The abscess formed in a strong and vigorous man who was attacked with pain in the right ear, fever, and finally, suppuration in the ear terminating in a discharge, in 1880. Six years later the patient consulted a specialist, who removed numerous polyps. Four years later he was attacked with pains in the temple, accompanied by vertigo and syncope from time to time. A year later the pains became more violent and the fever intense; finally these symptoms were followed by complete coma. When Terrillon first saw the case he diagnosticated an intra-cranial abscess near the petrous bone, probably between the dura mater and the bone. Trepanation was proposed and accepted, and the man, as already said, made a good recovery in the course of six months, and has remained well to the present time.—*Tribune Mèdicale*, 2d series, No. 3.

SUPPURATIVE OTITIS; CEREBRAL SYMPTOMS; TREPANATION; DEATH BY MENINGITIS; AUTOPSY.

DR. GELLÉ, in the *Tribune Médicale*, 2d series, No. 33, gives the history of a case of the nature above named occurring in a man forty years old. Pain was complained of in the left ear; no pain was felt in the mastoid process. Pain upon pressure was excited in the parotid region. There was violent headache, and there had been a chronic otorrhœa. Some relief was experienced by the opening of an abscess in the canal. The patient then left the hospital, returning, however, in eight days. The patient, after suffering intensely with headache and pains in the neck—without any pain, however, in the mastoid—with marked febrile symptoms, without high temperature, gradually sank into a coma in the course of three days. He was then trephined, but died two hours afterward without having recovered consciousness. The post-mortem examination revealed the existence of pachymeningitis throughout the extent of the sphenoidal fossa and in the occipital region, with sero-sanguinolent exudation around the petrous bone, and other results of intra-cranial inflammation.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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ARTHRITIS DEFORMANS OF THE LARYNX.

DR. W. E. CASSELBERRY, of Chicago, read a paper on this subject at the last meeting of the American Laryngological Association (*New York Med. Journ.*, vol. lviii., No. 16). He selects this term in preference to rheumatoid arthritis, in deference to his opinion that the disease is unrelated either to rheumatism or to gout.

A widow, fifty-eight years of age, had been afflicted for ten to fifteen years or more with progressive general symmetric arthritis of upper and lower extremities, but most markedly in the joints of the fingers and in the wrists. On two occasions, at intervals of two or three years, this patient had suffered with attacks of progressively increasing laryngeal dyspnœa similar to the third and last attack in which she came under the consultant care of Dr. Casselberry. She had then been suffering for some weeks with laryngeal dyspnœa which became so severe as to occasion marked inspiratory stridor and a notable expiratory noise.

The arytenoid eminences were swollen, but not grossly distorted, and were restricted in their movements. The posterior or cartilaginous portions of the vocal bands were swollen downward, upward, and inward, a condition attributed by the author to probable proliferative changes in the cartilaginous tissues. The abduction of the vocal cords, even in forced inspiration, was so slight as to leave but the smallest possible space between them free for respiration.

For two weeks the condition remained practically unchanged, and the debility from the dyspnœa became so great that an appointment had been made to perform tracheotomy, when respiration became somewhat easier and continued to improve until, at the end of six weeks, the patient breathed noiselessly and without conscious effort during quietude. Abduction was somewhat better, but the vocal bands could not be estimated to move more than one sixth of the proper distance, and there was no change in the aspect of the arytenoid eminences and the posterior extremities of the vocal bands. Treatment had been directly chiefly to relieve the dyspnœa and to reduce the congestion of the larynx, with alkaline and emollient sprays to free the parts from mucus and allay the inflammatory condition.

Abscess of the Larynx.

M. Gouguenheim has related (*Annales des Mal. de l'Oreille, etc.*, xix., 10) a case of tuberculous peri-laryngeal abscess proceeding from disease of the bodies of the lowest three cervical vertebræ, the sixth especially. Dyspnœa, progressive suffocation, cyanosis, and stridor rendered prompt tracheotomy necessary; but, although the trachea had not been incised, the operation gave issue to a large amount of pus with great amelioration for several days. The patient died after intense cervical, clavicular, thoracic, and brachial pains had existed for some fifteen days, with progressive paraplegia, retention of urine, and loss of reflexes.

Mr. Moll has reported (*Ibid.*) an intra-laryngeal abscess in a robust man, which appears to have been the first lesion in an attack of influenza with pleuro-pneumonia. The abscess underwent rupture in the vicinity of the vocal apophysis of the right vocal band, with subsidence of existing œdema of the right arytenoid cartilage, and restoration of mobility in the previously immobile vocal band.

Persistent Neuralgia of the Wing of the Nose.

M. Goris reports (*Annales des Mal. de l'Oreille, etc.*, t. xix., No. 10) a case of four years' duration developed at the close of a puerperal eclampsia. There

were facial paresis and difficulty in speech. Medicinal and electric treatment had failed. M. Goris elongated and resected the suborbital nerve in the cheek, with cessation of the pain, successfully maintained for the seven months that had since ensued.

Laryngitis Hiemalis.

Dr. J. C. Mulhall, of St. Louis, at the last meeting of the American Laryngological Association (*New York Med. Journ.*, vol. lviii., No. 16), reported a form of laryngitis occasionally observed by him during the preceding twelve winters; never during the intervening summers. He regards it as a winter variety of subacute catarrhal laryngitis in which the secretions are adhesive crusts from the beginning, producing dysphonia and even aphonia mechanically. Some of these patients would get laryngitis in summer, but there would be no crusts and no aphonia. Two of them rapidly lost all trace of the affection when transferred to the warm, moist air of Florida, and one of these had a recurrence on returning to St. Louis before cessation of the cold weather. It is not the ordinary laryngitis sicca and is not associated with similar disease of the pharynx or the nose, or necessarily with any other malady. Hygienic treatment, prophylactic and remedial, will often cure mild cases. Severe ones demand cleansing and then soothing sprays, repeated as required during the continuance of the cold season, while the internal administration of pilocarpine and ammonium chloride have been found serviceable.

Nasal and Rhino-pharyngeal Tumors.

At the Annual Reunion of the Society of Belgian Otologists and Laryngologists, June 4 (*Annales des Mal. de l'Oreille, etc.*, xix. 10), several interesting cases of fibroma and sarcoma were reported by Gevaert, Wagnier, Boval, and Natier.

Rhinitis Œdematosa.

Dr. J. C. Mulhall, of St. Louis, described to the American Laryngological Association (*New York Med. Journ.*, vol. lviii., No. 16) some cases of œdema of the mucous membrane of the turbinate bodies, in form somewhat resembling myxoma, and which he attributes to disease of the alimentary tract, chiefly of biliary origin, irritating the sympathetic nervous system. Intelligent dietetics, hydrotherapy, well-ordained physical exercise, massage, nerve rest, and the avoidance of drugs, form the basis of treatment. All topical treatment, except perhaps scarification, which affords temporary relief, should be avoided.

Amygdalith.

At the last Congress of Otologists and Laryngologists of Belgium, M. Lecocq presented (*Annales des Mal. de l'Oreille, etc.*, xix., 10) a calculus which he had extracted from a tonsil. It measured 26 millimetres in length and 13 in width, and weighed 2½ grammes.

Stricture of Larynx Several Years After Removal of a Neoplasm.

M. Gevaert (*Annales des Mal. de l'Oreille, etc.*, xix., 10) seven years ago removed with forceps a fibrous polyp from the larynx of a young girl. A year ago she presented herself with complete aphonia and pronounced stridor; both of about a year's duration. The vocal bands were found adherent by a sort of diaphragm which extended from the anterior angle to the posterior third of the glottic space, of which but a small oval aperture remained patent posteriorly. The membrane was incised with the concealed knife of Morell Mackenzie, and dilatation was maintained for eight days with the tracheal canulas of Stoerck. A slight readherence had been produced anteriorly.

Intubation of the Larynx..

Dr. Charles Whitney read a paper before the Surgical Section of Suffolk District Medical Society (*Boston Medical and Surgical Journal*, vol. cxxix., No. 14) based upon seventy-eight cases in his private practice. We note the following practical points: He has found some of the appliances furnished him defective, including gag, obturator, and even the tube; and he calls especial attention to the carelessness with which they are are often made. He has devised an obturator with an acuter angle than a right angle, and which he finds will render the introduction more easy than with the O'Dwyer angle. He assures himself that his tube is in position by inserting his finger into the œsophagus, and recognizing its anterior wall as a septum between the finger and the tube of the larynx. He also finds O'Dwyer's extractor defective, and recommends a better one, devised by Dr. Nichols, of the City Hospital, as so excellent as to avoid all trouble in extraction.

In the same journal (No. 16), Dr. George W. Gay, Visiting Surgeon of the Boston City Hospital, contributes a paper upon "Tracheotomy and Intubation" at the Boston City Hospital. There had been 456 tracheotomies with 110 recoveries, 24 per cent.; and 442 intubations without tracheotomy, with 90 recoveries, 20 per cent. In estimating their comparative merits both procedures should be studied, he justly contends, with reference to the amount of relief they give to the laryngeal dyspnœa; and on this ground he contends that with rare exceptions intubation affords as much relief as tracheotomy, while it can be practised in many cases in which the latter would be refused. Furthermore, he finds that section of the trachea after failure to relieve dyspnœa by intubation does not promise much benefit except in those cases in which membrane has been so pushed down by the tube that the patient is unable to expel it. At the same time the previous failure by intubation does not seem to lessen the chances of subsequent relief by tracheotomy, except in the gravest instances, when any disturbance may result in collapse.

In conclusion, he believes that intubation is preferable in the majority of cases in children under seven years of age.

[The views of Dr. Gay and of his colleague are doubly valuable from the fact that they have performed nearly as many intubations as tracheotomies, and both in great numbers; and that of late years intubations have been performed by them in by far the greater proportion.]

Recurrent Tonsillitis.

Dr. C. A. Leland strongly urges (*Boston Medical and Surgical Journal*, vol. cxxix., Nos. 15 and 16) division of the bridges between the walls of the diseased crypts, for which purpose he has devised a probed-pointed delicate knife-blade at an angle with its shaft. A number of interesting cases in point are detailed.

OBSTETRICS.

UNDER THE CHARGE OF

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Fatal Nausea and Vomiting of Pregnancy.

Davis (*Medical News*, June 2, 1894) at a meeting of the American Gynecological Society, May 29, 1894, presented a paper on the above subject, with a report of three cases, all with fatal termination.

Each of the patients presented as significant symptoms, a fatal condition of anæmia with profound alteration in the blood, as shown by purpuric spots more or less extensive; severe vomiting, accompained by straining, and in the later stages coffee-ground vomit; substernal pain; a temperature fluctuating a little above or below the norm, and a pulse of considerable rapidity.

A post-mortem on the second case revealed a most interesting condition: A longitudinal section of the uterus was made through the anterior wall. The organ was found to be dark purplish-red in color, its sinuses filled with soft currant-jelly like masses, not clotted. On the posterior wall, on a line extending between the orifices of the Fallopian tubes, was a transverse line resembling scar tissue. The internal os had been dilated, but was excessively resistant, the tissue resembling gristle. On the anterior and posterior walls of the cervix were areas of fibrous tissue, excessively firm, resembling that extending across the posterior surface. The stomach showed areas of thrombosis along its greater curvature. The heart was excessively friable and dark-red in color; valvular openings admitted two fingers; no evidence of atheroma could be found.

The blood was fluid, dark currant-jelly like in color, and without clots. No ante-mortem clot was found in the sinuses of the uterus.

A close examination of the uterus revealed an exceedingly interesting condition of the cervix; it consisted of dense connective tissue arranged in

whorls and containing two cysts, one an inch and a quarter in diameter, filled with yellowish fluid containing free small, ovoid, non-nucleated cells. A very interesting phenomenon shown in microscopical sections of the kidney was the presence of hyaline casts *in situ*.

No autopsies was made on the other two cases.

The author concludes that the danger of nausea and vomiting lies in the production of pernicious anæmia. When this is once established the uterus should be emptied. Replacing and retention by tampons suffice only for mild cases.

Migration of Ovum from the Ovary to the Tube.

Lodi (*Archiv für Gynäkologie*, 1893, B. 45, H. 2) after many experiments on rabbits, concerning the migration of the ovum from the ovary to the tube, summarizes his results as follows:

1. The infundibular and tubal cilia of the rabbit may be able to obstruct the passage of a body no larger than its ovum, provided the animal is at puberty.

2. The infundibulum is able, not merely to conduct an ovum from the ovary, but also from the free abdominal cavity into the inner genital tract. The old idea that the pavilion must apply itself to the ovary for the purpose of guiding the ovum is no longer held; a new experimental proof is afforded of the so-called external migration.

3. The migration of the ovum seems independent of the rut (in animals) and of coitus.

4. The first part of the journey through the tube is far more rapid than that near the uterus.

5. The tubal peristalsis seems to play but a small part in the migration, the small size of the ovum affords too small a point of effort, and also the direction of the peristaltic action varies. For picking the ovum out of the abdominal cavity, perhaps the firm lateral openings of the tube may avail by their ciliated margins.

New Treatment of Asphyxia Neonatorum.

Prochownick (*Centralblatt für Gynäkologie*, 1894, No. 10) describes a new mode of treatment of asphyxia neonatorum which he proposes as a substitute for Schultze's method. It consists in suspending the child by the feet, the head being lightly supported. If no assistant be available to hold the child's feet, the operator grasps the ankles between the fingers of his left hand. With the right he grasps the chest, the thumbs being in front, and makes six to eight regular compressions; at first expiration. The nose and mouth are freed from any mucus, which allows air to enter with the inspiratory effort. It may be necessary to douche the suspended child with warm water to assist the respiratory efforts.

The Induction of Labor in the Albuminuria of Pregnancy.

Puech (*Nouvelles Archives d'Obstétrique et de Gynécologie*, 1894, No. 3) contributes an article relative to the propriety of inducing labor in cases of gravid albuminuria. He considers the albuminuria to be regarded not

merely from the standpoint of its connection with eclampsia, but also to its bearing, especially if persistent, on the induction of permanent Bright's disease. Moreover, its influence on the fœtus is to be considered; as numerous observations seem to show that in cases of maternal albuminuria, the fœtus when born is frail or badly developed. Many die in utero and many abortions and premature labors are due to this cause. Post-mortem examinations reveal lesions of the placenta; hemorrhagic foci; atrophy of the villi or bloody effusions between the uterus and placenta. In the milk-diet we possess a means that surely places us beyond the reach of the great accidents of eclampsia, but this is not the sole consequence of albuminuria; the renal future of the mother must be considered, as also must the child threatened by placental hemorrhage. The various agents that act so well in non-puerperal albuminuria are ineffectual here until the pregnancy be interrupted, it being the cause of the condition. This not merely saves the mother, but in many cases the child as well. In a pregnant woman, if the albuminuria be persistent, accompanied by gastro-intestinal uræmia and dyspnœa, and if under a rigid milk-diet the albumin persists during eight or nine days, the maternal interests demand the induction of labor.

Symphysiotomy.

Müller (*Berliner klinische Wochenschrift*, 1893, No. 48) discusses the question of symphysiotomy, especially the partial section of the joint, as a partial substitute for the Cæsarean operation. He regards the new operation in a very favorable manner, and cites a case of a rhachitic V-para in whom labor was induced at thirty-two weeks for pelvic deformity. After rupture of the membranes it was found that the presentation had changed to a brow, the presenting part being firmly wedged in the inlet. After consultation, symphysiotomy was decided upon in preference to craniotomy and the Cæsarean operation, both of the latter having been considered. The operation was done by the external method, the symphysis being incompletely cut through. The child under strong pains was delivered spontaneously, but was so deeply asphyxiated that it was impossible to resuscitate it. The cord was twice encircling the neck. A portion of the abdominal wound was closed by silver-wire sutures, the remainder being filled with a tampon and left to granulate. No bone sutures were used. Three weeks later the patient was able to go about the room, but later was operated on for a vesico-vaginal fistula, which probably developed from pressure of the fœtal head during the considerable time preceding the operation in which the patient was in labor.

Puerperal Inflammation.

Dolèris (*Nouvelles Archives d'Obstétrique et de Gynécologie*, 1894, No. 3) contributes an article relative to puerperal inflammation. He concludes:

1. That puerperal inflammation is derived from a streptococcus identical with the streptococcus pyogenes.

2. That suppurative inflammations exist, of less intensity, in which the staphylococcus plays the principal part.

3. In the pus of certain cases of peritonitis and of puerperal lymphangitis

following gangrenes of the uterine mucous membrane, vibriones and bacilli of putrefaction may be found. The habitat of the streptococcus is the surface about the point of the placental attachment, the clots that may have formed in the orifices of the sinuses, and any discharges that may be in the uterine cavity; also in pus, though if this be in a closed cavity the streptococcus soon degenerates or dies. The pathogenic action of the streptococcus may be—

1. Hypertoxic, by its toxines, and limited to the first stage of the inflammation.
2. Destruction of vitality, and a cause of necrobiosis *en masse* of the tissues.
3. Pyogenic or septic at the same time. The vitality of the streptococcus is uncertain; the author believes it may lie dormant in the tissues for years.

There are three leading types of this inflammation:

1. Inflammation and local suppuration.
2. Inflammation and migratory suppuration.
3. Hypertoxic infections—non-suppurative.

The first group represents nearly always the mildest complications of recent puerperal traumatism, be they slight or grave.

In the second group, by far the most common, is the mixed migration of the streptococcus. It follows the mucous passages by means of the lymphatics to the pavilion of the tube and the peritoneum, also along the network of intra muscular interspaces, perforating thus the uterine walls, from mucous membrane to the peritoneum, the parametrium, the ovary, or the tube.

The Causation of Spondylolisthesis.

Neugebauer (*Zeitschrift für Geburtshülfe und Gynäkologie*, Band xxvii., Heft 2) contributes an article upon the causes producing forward displacements of the vertebra (spondylolisthesis), and the differential diagnosis of the same. The author narrates several cases, and gives numerous well-executed illustrations of instances where the condition was the result of accident or injury in girlhood or adult life; the result of the injury being the throwing forward of the fourth or fifth lumbar vertebra, with the formation of a sharp forward bend at the point of the sacro-lumbar union. The patients presented a marked folding of the abdominal walls, giving the body a telescoped appearance. In other cases, the body at the lumbo-sacral articulation made an abrupt forward bend, the abdomen being enormously pendulous, espcially in pregnancy. In many other cases narrated, marked rhachitis existed and was the cause of the spinal bend.

The author also alludes to the published cases and opinions of Lane, relative to women who have performed hard labor in carrying weights, where in some cases the lower lumbar vertebræ were forced forward. In these cases there may be destruction of the bodies and intra-vertebral substance from the tenth dorsal to the second lumbar, especially in front. The bodies of the third, fourth, and fifth lumbar vertebræ, with their fibro-cartilages, as they form part of the convexity of an antero-posterior curve, will be but little diminished in front. The force tends to displace the lumbar vertebræ forward and downward, the fifth lumbar being pushed forward, projecting beyond the sacrum. Neugebauer regards this as something quite peculiar and local, while Lane considers it as a part of a general change in the spinal column, very often due to the vocation.

GYNECOLOGY.

UNDER THE CHARGE OF

HENRY C. COE, M.D., M.R.C.S.,
OF NEW YORK.

Perforation of the Uterus during Curettage.

Alberti reported at a recent meeting of the Berlin Obstetrical Society (*Centralbl. für Gyn.*, 1894, No. 21) the case of a patient, aged thirty-two years, who had borne four children. Her menstruation had long been profuse, when, after skipping a period, she began to have increased flow and a foul discharge. She was very anæmic and had elevation of temperature. Her physician performed curettage, using a sharp instrument; he then introduced polypus forceps in order to remove supposed decidual membrane and drew out a loop of intestine. He pushed the intestine back into the vagina, tamponed with gauze, and sent the patient to the hospital, where Alberti performed cœliotomy three hours after the accident. A perforation an inch long was found on the right side of the fundus uteri, through which a loop of gut was prolapsed. It was so firmly grasped at the os internum that it was necessary to use a hernia-knife in order to free it. The prolapsed portion (seven inches in length) was much congested and was black in color at the point of constriction. The uterine wall was unusually thin at the site of the perforation, which was closed with four Lembert sutures. The patient had a normal convalescence, but four weeks after the operation the hemorrhage recurred, so that it was necessary to repeat the curettage, a quantity of ordinary fungosities being removed. Six weeks later the same operation was repeated because of renewed bleeding. Two years had since elapsed and the patient had remained perfectly well.

In the discussion which followed Veit reported a similar case in which he recognized the seat of the perforation on the posterior uterine wall by examination per rectum, opened Douglas's pouch, and succeeded in replacing the gut after enlarging the tear. As there was considerable hemorrhage and the uterus was probably septic, he performed vaginal hysterectomy, but the patient died of peritonitis. He regarded the forceps as a dangerous instrument in cases of abortion. A soft uterus could be perforated even with a sound; it made little difference whether a curette was sharp or dull, since the perforation was doubtless made with the rounded end rather than with the edge, but when the forceps was used there was the additional danger that a loop of intestine might be drawn through the opening. He was opposed to the removal of retained products of conception with forceps, unless they were first located with the forefinger and the forceps introduced along it as a guide.

Gusserow cited a case which he had seen in consultation in which the omentum had been pulled down with forceps. He opened the abdomen, which contained a considerable quantity of blood, ligated and excised the

prolapsed portion of omentum, and amputated the uterus, which was presumably septic. The patient had practically recovered from the operation, when she died suddenly from pulmonary embolism on the twenty-fifth day.

ORTHMANN stated that he had been called to see a patient by her physician, who had perforated the uterus with forceps and badly lacerated a loop of intestine, tearing away its mesentery. On performing cœliotomy he found the intestine was adherent to the fundus uteri at the seat of the perforation. The uterus and adnexa were amputated and the injured portion of gut was resected, the patient making a good recovery.

OLSHAUSEN had observed a similar case, in which he was obliged to resect the intestine, but the patient was already septic and succumbed soon after. He was opposed to the use of both the sharp curette and the forceps in these cases; in fact, he employed instruments just as little as possible, preferring to remove foreign material with his finger.

MARTIN took a somewhat different position from the other speakers. He had seen a case in which an experienced gynecologist had perforated the uterus with forceps and drawn down thirty inches of intestine. Nothing could be done, as the patient was already moribund. In three cases of curettage out of several thousand in his own practice he had three times perforated the uterus with forceps, and once grasped a loop of intestine, but immediately recognized the fact and released the gut, when it slipped back into the abdominal cavity. Other surgeons had had a similar experience. All the patients had recovered without any reaction. Personally, he was unwilling to reject the forceps in cases of abortion, since he had used it satisfactorily for twenty years and could not dispense with it. When the cervix was sufficiently dilated to admit the finger, they were, of course, unnecessary. In cases in which it was necessary to first dilate the os, he used the forceps to remove fragments which had been detached with the curette or could not be removed with the fingers.

OLSHAUSEN strongly disapproved of Martin's recommendation of the forceps in incomplete abortions before the fourth month. In very early cases particularly, it was a dangerous instrument, and only the finger or curette should be employed. While an unskilful operator might do serious damage, even with his finger, he was much more likely to do so with the forceps.

VEIT agreed with the last speaker, and criticised Martin's statement that one could recognize when he had seized intestine and could release it from the grasp of the forceps without injury. Such skill would be possessed by few, and it was certainly wise to warn the general profession that this instrument should never be inserted into the uterine cavity except under the guidance of the finger.

[We have ventured to introduce this discussion *in extenso* because it deals with a subject of vital importance. The curette-forceps, devised by Dr. Emmet, is an exceedingly useful instrument for removing retained products of conception, but we are personally cognizant of two cases in which it was thrust through the uterine wall and a loop of intestine was drawn from the vulva. Cœliotomy was performed in both instances, one case (reported by Dr. G. W. Bratenahl) being successful. Doubtless other unreported cases have occurred in this country, as the instrument is a favorite one with some operators. The advice to use forceps only as an adjunct to the finger and curette deserves general acceptance.—H. C. C.]

Carcinoma Uteri.

Müller (*Ibid.*) summarizes the results of his observations in 577 cases treated in Gusserow's clinic. In over a third of the number the patients were under the age of forty. There were none under twenty, the average being between thirty-five and forty. Vaginal extirpation was performed in 15.4 per cent., of which 52.92 per cent. of the patients were under forty. Forty per cent. of the whole number observed had passed the menopause, 60.1 per cent. of the latter being over fifty; 5.3 per cent. of the patients were nulliparæ. Contrary to the accepted belief, there was no direct relation between the number of children and the frequency of the disease, though the average number of abortions was high—0.57 per cent. apiece. In 8.14 per cent. the disease developed during pregnancy or the puerperium.

In some cases, especially inoperable, the initial symptoms were entirely absent, early pain being the exception. Bleeding during coitus was especially frequent. Menorrhagia and metrorrhagia were usually the first symptoms observed, foul discharges being later evidences. Vesical and rectal irritation were not infrequently noted. The duration of the disease appeared to be longer in old than in young subjects.

Fixation of a Wandering Spleen.

Richelot (*Gaz. des Hôpitaux; Centralblatt für Gyn.*, 1894, No. 21) examined a patient with a painful tumor in the right iliac fossa, which was thought to be of appéndiceal origin, or possibly tuberculous. On opening the abdomen it was found to be a displaced liver, adherent to the abdominal wall. The adhesions were separated, the organ was replaced, and was fixed in position with catgut sutures passed through the capsule and the parietal peritoneum. Three months after the operation the liver was found to be in its normal position, and the patient was entirely relieved of his former symptoms—pain and emaciation.

Corrigendum.—On page 81 of The Journal for July, 1894, in the review of Forselles' *Thrombosis of the Lateral Sinus*, the original German title was by accident omitted. Below will be found the German title:

"Die durch eitrige Mittelohrentzündung verursachte Lateral-Sinus Thrombose, und deren operative Behandlung. Von Arthur af Forselles, Assistenzarzt der chirurgischen Klinik zu Helsingfors."

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